Vol. 43—No. 202
10-18-78
BOOK 2:
Pages
48299–48617

federal register

BOOK 2 OF 2 BOOKS

WEDNESDAY, OCTOBER 18, 1978
PART II—Continued

ENVIRONMENTAL PROTECTION AGENCY

■

WOOD PRESERVATIVE PESTICIDES

Initiation of Schedule for Review

and

Notices of Rebuttable Presumption Against Registration of Certain Pesticides

in more than 90% of the samples. Arsenic levels ranged up to 3.40 mg/kg in 1971, 1.70 mg/kg in 1972, and 1.24 mg/kg in 1973. Fish from the Great Lakes had high residues more frequently than fish from other stations.

(5) Birds

Martin and Nickerson (1973) conducted a nationwide study of arsenic and heavy metal residues in starlings (*Sturnus vulgaris*), which were collected in 1971 at 50 sites throughout the continental United States. The authors noted that arsenic residues were low (<0.04 ppm). Only one sample from Michigan showed a higher arsenic content (0.21 ppm). Eight samples showed no detectable arsenic resiudes.

White et al. (1977) conducted a similar study in starlings, which were collected in 1973 at 51 sites throughout the continental United States. The authors noted that residue levels were generally low; arsenic residues ranged from <0.05 to 1.40 ppm. The average level for all 51 sites was 0.160 ppm. The authors also stated that there had been a significant increase in arsenic residues in starlings since 1971. Arsenic residues were 88% higher in 1973. Twenty-eight of 1973 samples (55%) contained residues of 0.10 ppm or greater.

Blus et al. (1977) studied residues of organochlorine pesticides and 10 heavy metals in livers and eggs of brown pelicans (*Pelecanus occidentalis*), a rare and endangered species, from 1969 through 1973. Pelican colonies in Florida (1969 to 1970) and South Carolina (1971 to 1972) were sampled. Arsenic residues in eggs from South Carolina were three times higher than those from Florida. Arsenic residues in eggs from South Carolina and Florida, respectively, ranged from 0.076 to 0.85 ug/g and 0.07 to 0.18 ug/g fresh wet weight.

Livers of four brown pelicans, shot in 1970, were checked for arsenic residues. Livers of an adult female and an immature male, both shot in Florida, contained 0.63 and 0.89 ug of arsenic/g fresh wet weight, respectively. Livers of a 6-week-old female and an adult male, both shot in South Carolina, contained 0.36 and 0.29 ug/g, respectively.

Livers of four brown pelicans, found dead in 1972 and 1973, were also checked for arsenic residues, with the following results: 0.47 ug arsenic/g fresh wet weight in an adult female found in Florida in 1972; 0.23 ug/g in an adult female found in Georgia in 1972; 0.54 in an immature male found in Florida in 1973; and 1.02 ug/g in an 8-week-old female found in South Carolina in 1973.

The authors noted that each egg and tissue sample of brown pelicans that was analyzed contained residues of ten heavy metals, but that the significance of these residues to the pelicans was undetermined.

Lynch (1973) studied the occurrence of selected toxic metals, including arsenic, in pheasants (Phasianus colchicus) in Ohio. Twenty-six samples of pheasant breast muscle were analyzed for arsenic with a detection limit of 0.001 ppm. Sixteen samples contained detectable arsenic residues. The highest amount found was 0.172 ppm. The author indicated that, although lead arsenate was recommended for use in apple orchards in 1973, only 1% of Ohio orchards were treated with it. The author did not state whether arsenic residues in pheasants resulted from natural background levels or from agricultural or industrial pollution.

(6) Mammalian Wildlife

Lynch (1973) studied the occurrence of selected toxic metals in cottontail rabbits (Sylvilagus floridanus),

squirrels (Sciurus carolinensis and S. niger), and white-tailed deer (Odocoileus virginianus) in Ohio. The detection limit for arsenic residues was 0.001 ppm. Eleven samples of deer leg muscles were analyzed; six contained detectable arsenic residues. The highest amount found was 0.217 ppm.

Three samples of rabbit femur were analyzed, but none contained detectable arsenic residues. Twenty-two samples of rabbit leg muscle were also anlyzed; four contained detectable arsenic residues. The highest amount found was 0.020 ppm. A single sample of squirrel brain contained 0.055 ppm arsenic. Of four samples of squirrel femur analyzed, one contained arsenic at 0.017 ppm. A single sample of squirrel liver contained 0.035 ppm arsenic. Twenty-three samples of squirrel leg muscle were analyzed; 13 contained detectable arsenic residues. The highest amount found was 0.035 ppm.

(7) Food and Feed

The following studies indicate that inorganic arsenic residues (as As_2O_3) in various foods are generally below the established tolerance levels. Arsenic occurs in food and feed as either a natural constituent or as a residue from pesticides or smelter pollution.

Generally, the only foods which are high in arsenic are seafoods. Schroeder and Balassa (1966) sampled food and

beverages available in grocery stores and found that fish and other seafoods had the highest levels (mean of 4.64 ppm) while fruits had the lowest content (mean of 0.042 ppm).

Chapman (1926) reported mean values of up to 80 ppm of arsenic in mussels, oysters, and scallops, while mixed fresh water fish contained 0.65 ppm.

Bradicich et al. (1969) studied arsenic residues in samples of various food commodities in Texas in 1963 from areas where arsenical defoliation had been practiced. The study was continued in 1964, 1965, and 1966 for cottonseed products. A majority of the samples for 1963 (77 out of 107) showed arsenic residues; the average residue levels ranged from 0.01 to 0.90 ppm, and the maximum residue levels ranged from 0.01 to 3.72 ppm. Over 95% of the cottonseed oil and cottonseed meal samples showed residues significantly higher than the other commodities.

Of the 159 cottonseed-product samples tested from 1963 through 1966, 143 were positive, but only three were above the established tolerance level of 4 ppm for As_2O_3 (see Section I.D). The average residue level for all hull samples was 1.2 ppm, for all meal samples 1.4 ppm, and for oil samples (the main product of human consumption) 0.4 ppm.

In 1970 Steevens et al. (1972a) studied arsenic levels in flesh and peelings of potato tubers grown in Wisconsin potato fields on which sodium arsenite had been extensively used as a defoliant from 1958 to 1968. (The use of sodium arsenite as a potato vine defoliant was cancelled in 1969.) The arsenic concentration in potato peelings ranged from 0.2 to 2.3 ppm and in potato flesh ranged from a trace to 0.6 ppm. The arsenic (As) tolerance limit at that time was 2.6 ppm.

Crecelius (1977a) analyzed 19 samples (plus one duplicate of each of two samples) of domestic table wines from several types of arsenic (arsenite, arsenate, methylarsonic acid [MAA], and dimethylarsinic acid [DMAA]). Arsenite concentration in these samples ranged from < 1 to 420 ppb, and arsenate from < 1 to 110 ppb. Both MAA and DMAA levels were below the detection limit of 1 ppb in these samples. The United States does not have an arsenic standard for wine, although a maximum value of 20 ppb was once recommended. Great Britain's maximum tolerable arsenic level in wine is 100 ppb, and several other countries (not identified by the author) have a limit of 200 ppb. The maximum arsenic level for drinking water in the United States is 50 ppb. The proposed 20 ppb U.S. Standard for wine was exceeded by 15 of the 19 samples for arsenite and 14 samples for arsenate; the 50 ppb U.S. water standard was exceeded by 12 samples for arsenite and two samples for arsenate; the 100 ppb British

Standard for wine was exceeded by ten samples for arsenite and one sample for arsenate; the 200 ppb standard from "other countries" for wine was exceeded by five samples for arsenite. The author did not investigate agricultural practices in the vineyards. The author described the arsenic levels in some samples as "surprisingly high" and suggested they were due to use of arsenicals in the production of grapes. The tolerance level for lead arsenate on grapes is 7 ppm and for sodium arsenate is 3.5 ppm. There is an interim tolerance of 0.5 ppm for use of sodium arsenite on grapes.

The Food and Drug Administration (FDA) conducts periodic Market Basket Surveys of pesticide residues in total diet samples of ready-to-eat food. The samples are representative of a two-week intake for males 16 to 19 years old from moderate income families. Composite samples of food commodity groups are analyzed for arsenic as As_2O_3. The analytical method does not distinguish between naturally occurring arsenic and arsenic residues from industrial or agricultural sources. The following studies reported on arsenic residues found in FDA's Market Basket Survey.

Cummings (1966) reported that arsenic residues in samples from Baltimore, Maryland, analyzed in May 1964,

ranged from 0.10 to 4.7 ppm. Food commodity groups in which arsenic residues were found were meat, fish, and poultry; grain and cereal products; potatoes; legume vegetables; root vegetables; and fruit.

Duggan et al. (1966) reported on pesticide residues in diet samples collected bimonthly from June 1964 through April 1965 from retail food stores in Boston, Massachusetts; Kansas City, Missouri; and Los Angeles, California. Arsenic (As_2O_3) was found in six of the 216 composite samples assayed, at levels ranging from 0.1 to 4.7 ppm. Arsenic was present in one of the 18 meat, fish, and poultry samples at 0.12 ppm; in one of the 18 grain and cereal samples at 0.10 ppm; in one of the 18 potato samples at 4.7 ppm; in one of the 18 legume vegetable samples at 0.11 ppm; in one of the 18 root vegetable samples at 0.10 ppm; and in one of the 18 fruit samples at 0.18 ppm.

Duggan and Weatherwax (1967) reported on the average daily intake of all pesticide residues in or on the total diet samples collected in 25 cities from June 1964 through April 1966. The results showed that the daily intake of arsenic residues (As_2O_3), expressed in milligrams per day, for samples collected from June 1964 through April 1965, were trace (>0.001 mg) for meat, fish, and poultry samples; 0.002 for grains and cereals; 0.063 for potatoes; 0.001 for legume vegetables; 0.001 for root vegetables; and 0.002 for fruits.

Results for samples collected from June 1965 through April 1966 were 0.003 for meat, fish, and poultry; 0.001 for grain and cereals; trace for root vegetables; 0.001 for garden fruits; and 0.001 for sugars and adjuncts. The authors noted that whereas the average 16-to-19-year-old male consumes about 8.8 pounds of food and drink daily, the average individual in the general population consumes only about 4.8 pounds daily.

Duggan et al. (1967) reported on pesticide residues in total diet samples collected from June 1965 through April 1966. Arsenic residues, ranging from 0.1 to 0.5 ppm (As_2O_3), were found in 5 of 28 composites of meat, fish, and poultry; in one of 28 composites of grain and cereals at 0.1 ppm; in one of 28 composites of root vegetables at 0.1 ppm; in one composite of garden fruits at 0.1 ppm; and in one of 28 composites of sugars and adjuncts at 0.1 ppm.

Martin and Duggan (1968) reported on 360 total diet samples, from 30 markets in 29 cities for June 1966 through April 1967. Arsenic (As_2O_3) was found in 33 diet samples; residue levels ranged from 0.1 to 0.4 ppm. Eight samples showed arsenic residues below the analytical sensitivity level.

H. Pesticide Episode Reports System (PERS)

EPA's Pesticide Program maintains a Pesticide Episode Reports System (PERS) which collects reports of pesticide exposure affecting humans, domestic animals, livestock, and wildlife (EPA 1977). According to those records, there were 308 episodes from 1966 through April 1977 involving various inorganic arsenicals.

There were 25 episodes involving use of arsenic trioxide alone as a rodenticide or insecticide. All of these involved humans. For 15 of these 25 episodes, which include seven fatalities, there was substantial evidence that arsenic trioxide was directly involved; and for eight episodes, there was insufficient evidence to either prove or disprove involvement of arsenic trioxide.

There were 17 episodes involving use of arsenic trioxide as an herbicide. Seven of these involved other active ingredients, including arsenic acid, lead arsenate, arsenous oxide, heptachlor, and sodium chlorate. Six of these 17 episodes involved livestock, five involved crop contamination, three involved humans, and one each involved a bee kill and water contamination. For seven of these episodes which include one fatality, there was substantial evidence that arsenic trioxide was directly involved; for

five episodes, there was insufficient evidence; and for one episode, there was evidence which proved that arsenic trioxide was not responsible.

There were 128 episodes involving use of sodium arsenate and/or arsenite as an insecticide and wood preservative. One of these episodes involved other active ingredients. One of these episodes involved a dog, one involved livestock, and the other 126 episodes involved humans, mostly children. For 14 of these 128 episodes which include two fatalities, there was substantial evidence that sodium arsenate/arsenite was directly involved; for 16 episodes which include one fatality, there was circumstantial evidence; and for 98 episodes which include one fatality, there was insufficient evidence to either prove or disprove involvement of sodium arsenate/arsenite.

There were 50 episodes involving use of sodium arsenite as an herbicide. In three of these episodes other forms of arsenic were also involved. One each of the 49 episodes involved a stand of trees and a dog; 20 involved livestock; and 28 involved humans. For 34 of these episodes which include twenty fatalities, there was substantial evidence that sodium arsenite was directly involved; for five episodes, there was circumstantial evidence; for 10 episodes which include one fatality, there was insufficient evidence; and for one episode, there was evidence which proved that sodium arsenite was not responsible.

There were 33 episodes involving use of lead arsenate as an insecticide. Boric acid was involved in two of these episodes, of which 31 involved humans, one involved livestock, and one involved contamination of well water. For five of these 33 episodes, there was substantial evidence that lead arsenate was directly involved; and for 28 episodes, there was insufficient evidence to either prove or disprove involvement of lead arsenate. There were no fatalities in any of the episodes involving humans.

There were 16 episodes involving use of calcium arsenate as an insecticide or herbicide. Five of these involved calcium arsenate alone, and eleven also involved other active ingredients (mostly metaldehyde). Twelve of these episodes involved humans, two involved livestock, and one each involved a dog and well water contamination. For three of these 13 episodes, there was substantial evidence that calcium arsenate was directly involved; for four episodes, there was circumstantial evidence; for eight episodes which include one fatality, there was evidence which proved that calcium arsenate was not responsible.

There were three episodes involving use of copper acetoarsenite as an insecticide, all of which involved humans.[4/] For one episode, there was substantial evidence

4/ In one episode, other active ingredients (MSMA, diazinon, and malathion) were also involved.

that copper acetoarsenite was directly involved; and for two episodes, there was insufficient evidence to either prove or disprove involvement of copper acetoarsenite. No fatalities were reported.

In addition, 36 episodes involving various inorganic arsenicals were reported which were not use-related. These episodes involved suicides and homicides, fires, and transportation accidents.

I. Lead Arsenate - Chronic Effects Due to Lead

Although lead arsenate contains a lead moiety, the chronic effects seen in vintners and others who use the compound do not correspond to chronic lead effects. Rather the effects are typical of those seen with other inorganic arsenicals (see Section IV. B). In the human body, inorganic lead has damaging effects on the hematological, neurological, and renal systems (Nordberg 1976). The Agency will conduct a separate evaluation of possible adverse effects due to lead in the future.

Lead interrupts many steps in the pathway of heme synthesis and formation of hemaglobin, both part of the blood-forming process. Lead also shortens the life span of erythrocytes. Lead affects both the central and peripheral nervous systems. Encephalopathy is a major effect on the central nervous system and

usually results from a more intensive and prolonged exposure to lead than that inducing hematological effects. The acute form may be fatal. Peripheral nervous system disorders due to chronic lead exposure are characterized by selective involvement of motor neurons with little sensory involvement. Lead effects on the renal system, such as nephritis, are seen primarily in adults with heavy occupational exposure. The long-term effect on the kidneys is nonspecific nephropathy.

Teratogenic and reproductive effects of lead in humans have not been studied extensively. There are reports of steriltiy, stillbirths, and miscarriages in women industrially exposed.

J. Arsenate and Arsenite: Interconversion

Studies discussed in this portion of the position document indicate arsenate (in either the organic or inorganic form) to be the predominant form of arsenic in mammals. When inorganic arsenite is administered the majority of the arsenic is converted (oxidized) to the less acutely toxic pentavalent form. Information on the conversion of the less acutely toxic pentavalent arsenicals (arsenates) to the more acutely toxic trivalent form (arsenites) is of importance in evaluating the environmental and health effects of the inorganic arsenic compounds used as pesticides.

Based on studies presented in this section and on an analysis by EPA's Carcinogen Assessment Group (EPA 1978) the Agency concludes that although oxidation of trivalent arsenic (arsenite) to the pentavalent form (arsenate) is the favored reaction, pentavalent arsenic (arsenate) can be reduced in vivo to the trivalent form (arsenite). This conclusion is supported by: 1) in vivo experiments in mammals, 2) qualitative similarities in toxic effects of arsenite and arsenate, and 3) data showing an equilibrium ratio between arsenate and arsenite. For additional information dealing with metabolism see Sections I.E(1)(c), (d), and I.E.(5).

(1) Metabolism - In Vivo Interconversion

The following studies show that inorganic arsenite (trivalent) is oxidized in vivo to arsenate (pentavalent) and/or that inorganic arsenate is reduced in vivo to arsenite.

Mealey et al. (1959) demonstrated that conversion takes place within human body tissues. Radioactive inorganic arsenite was administered intravenously to five humans; the amounts of arsenite and arsenate in the urine were monitored over time. During the first four days, the percentage of

arsenite to arsenate in the urine leveled off at about 75% arsenate. Results indicated that oxidation of arsenite to arsenate took place within body tissues.

Winkler (1962) had findings similar to Mealey et al. (1959) after identifying and estimating arsenic residues in livers of rats ingesting arsenicals. Rats were fed sodium arsenite and sodium arsenate at 125 and 250 ppm of arsenic. The animals were killed after 90 days, and their livers were analyzed. The results showed that trivalent inorganic arsenic was largely changed to the pentavalent form in the rat. Pentavalent arsenic, however, was not reduced to the trivalent form.

Lakso and Peoples (1975) observed essentially similar levels of methylated arsenic and inorganic arsenic (individual species not measured) after feeding experiments with cows and dogs in which both sodium arsenate and potassium arsenite were administered. The authors suggested that the short delay in increased methylated arsenic excretion after arsenite feeding is indicative of the oxidation of arsenite to arsenate prior to methylation.

Crecelius (1977b) studied changes in the chemical speciation of arsenic following ingestion by man. The

subject of this study was one 30-year-old male whose diet was controlled to minimize the intake of arsenic from any other food or drink before and during the experiment.

In the experiment, 450 ml of wine, containing 50 ug trivalent and 13 ug pentavalent arsenic, was ingested. The major form of arsenic excreted in the urine after 61 hours was dimethylarsinic acid (DMAA), a pentavalent arsenical, which accounted for about 50% of the total 63 ug of arsenic ingested. Trivalent and pentavalent arsenic each accounted for 8%, and methylarsonic acid (MAA), another pentavalent arsenical, for 14%. The author concluded that, when trivalent arsenic is ingested, a small portion is excreted unchanged as arsenite, but that the majority is oxidized and methylated in the body and excreted as DMAA and MAA (pentavalent arsenic).

Conversion of the trivalent to the pentavalent form of arsenic has often been described in the literature (Oak Ridge 1976; EPA 1976b; AWPI 1977).

Although the in vivo oxidation of trivalent arsenic to pentavalent arsenic is the favored reaction, the possibility of reduction in vivo of pentavalent arsenic to the trivalent form has been suggested by several investigations.

Thuret (1939) reported that in vitro experiments showed reduction of pentavalent arsenic, in contact with

liver pulp, to the trivalent form after six hours. Lanz et al. (1950), in a study on rats using radio-labeled sodium arsenate, reported that 10 to 15% of the inorganic arsenic recovered from the urine was in the trivalent form.

Ginsburg and Lotspeich (1963) and Ginsburg (1965) observed that arsenates are reduced to arsenites under certain conditions. These studies are of particular concern to the Agency in that they report reduction in vivo.

Ginsburg and Lotspeich (1963), in a study on the interrelation of arsenate and phosphate transport in the dog kidney, reported in vivo reduction of arsenate to arsenite during intravenous infusion of a solution containing pentavalent arsenic and 1.5% trivalent arsenic. About 5.5% of the plasma arsenic and 14% of the urinary arsenic was in the trivalent state during the first three periods of the experiment. The authors also noted a net secretion of trivalent arsenic in the urine because of intracellular reduction by renal tissue.

In further investigations, Ginsburg (1965) demonstrated that some infused arsenate is reabsorbed by the dog kidney and reduced to arsenite. A representative experiment measured the kidney clearance of arsenate and arsenite during the intravenous infusion

of a solution containing arsenate (98.6%) and only 1.4% arsenite. If both valence forms are treated similarly by the kidney (i.e., no conversion), the relative proportions of pentavalent and trivalent arsenic in the urine should be the same as in the plasma. After 35 minutes of infusion, however, 5.6% of the plasma arsenic and 14% of the urine arsenic was trivalent. Based on comparisons with in vitro experiments, this higher level of arsenite in vivo was not accounted for by simple filtration by the kidney or by a greater reduction of arsenate in the urine than in the plasma. In 15 control experiments the ratio of the percent of arsenite in urine to the percent of arsenite in plasma ranged from 1.9 to 4.2 (mean 2.8). After infusion of arsenate, there was a net renal tubular reabsorption of arsenate (26% of the filtered arsenate) and a net secretion of arsenite occurred. The rate of arsenite secretion was directly proportional to the rate of arsenate reabsorption. There was also a greater concentration of arsenite in renal venous plasma than in renal arterial plasma.

In another experiment where a solution of arsenite (91.6%) containing 8.4% arsenate was infused into the dog, arsenate appeared in the plasma and urine

at levels greater than 8.4%, indicating in vivo oxidation. There was a net reabsorption of trivalent and, to a lesser extent, of pentavalent arsenic. There was no net secretion into the urine of arsenite during this infusion which indicates that arsenite is probably not actively transported in the kidney. When arsenate was then added to the infusion solution in the second phase of the experiment, the additional arsenate was available for reabsorption. Consequently, the former net reabsorption of arsenite changed to a net secretion of arsenite, indicating a conversion of arsenate to arsenite.

After experimentally examining several hypothetical explanations for the increased presence of arsenite in the urine and plasma, the author concluded that arsenite is secreted into urine and blood from an intracellular site (tubular cells) in the kidney, where some of the arsenate reabsorbed from the glomerular filtrate is intracellularly reduced to arsenite. This process is dependent on a concentration gradient for arsenite from cell to urine which is established by the intracellular reduction of the arsenate reabsorbed from the glomerular filtrate. Ginsburg noted that this gradient for arsenite from cell to urine and blood was established only when the arsenic infusion contained large amounts

of arsenate relative to arsenite. He also noted that it is not known whether the arsenic retained in the kidney cells is in the form of arsenate or arsenite.

Peoples (1964), however, failed to show in vivo reduction of arsenate to arsenite. In this study arsenic acid (a pentavalent arsenical) was fed to lactating Holstein cows at levels of 0 to 1.25 mg/kg daily for eight weeks. The results showed no significant variation in weekly milk samples at any feeding level from controls, although small amounts of arsenic acid were detected. The arsenic found in the livers of two cows fed arsenic acid was entirely in the pentavalent state. The author concluded that the small amounts of tissue-bound arsenic were entirely in the pentavalent form and that there was no evidence that it was partly reduced to the trivalent form.

The Occupational Safety and Health Administration [OSHA (1978)], has concluded "that the presently available evidence indicates that there is little or no conversion of pentavalent to trivalent arsenic in

the body." In reaching this conclusion OSHA relied heavily on testimony by Dr. Peoples (1975) during hearings held by OSHA on inorganic arsenic. Dr. Peoples concluded that the Ginsburg findings "could not be relied on because the analytical method was not accurate in the range of the reported sample size."

In an effort to answer the question of the realiability of the studies by Ginsburg and Lotspeich (1963) and Ginsburg (1965), EPA submitted these studies for outside review. In validation reports for the Agency on both Ginsburg papers, Lombardini (1978b; 1978c) concludes that Ginsburg's observation of in vivo reduction of arsenate to arsenite were supported by the data given. In addition, Lombardini (1978a) evaluated the paper by Crawford and Storey (1944) which described the analytical method used by Ginsburg. Lombardini stated that the method was "somewhat of a classical technique...still in use in 1978." He added, "This procedure has been time-tested and is valid."

Although the issue is not free from doubt, the Agency concludes that sufficient scientific information is available to support the conclusion that

pentavalent arsenic can be converted to trivalent arsenic in the body. In questions such as this one on the frontiers of scientific knowledge, decision-making must to a large extent rest on policy judgments. In view of the scientific information available and the serious health issues at stake, the Agency has made the judgment that it is in the public interest to bring forward through the RPAR process its tentative conclusion that pentavalent arsenic converts to trivalent arsenic *in vivo*. The RPAR process is intended to be an information-gathering process which will provide the broadest base possible for final regulatory decision-making.

(2) Toxicity - Qualitative Similarities and Quantitative Differences

The similarity of the toxic effects of trivalent and pentavalent arsenicals may provide additional support for the proposition that there is *in vivo* interconversion of arsenate to arsenite. The following studies describe some of the qualitative similarities and the quantitative differences in the effects of arsenate and arsenite.

The acute toxicity (via intraperitioneal injections) of sodium arsenite to rats is about 3.6 times that of sodium arsenate (Franke and Moxon 1936).

In a teratology study of random-bred albino Swiss-Webster mice, Hood (1972) administered sodium arsenite via single intraperitoneal injections on days 7 to 12 of gestation at dose levels of 10 and 12 mg/kg. He observed the same spectrum of abnormalities which he had seen with a 4.5 times higher sodium arsenate treatment of 45 mg/kg (Hood and Bishop 1972). The incidence of defects was slightly higher with the 45 mg/kg arsenate. Although the author admits that the similarity of abnormalities does not prove the mechanism of action is identical for the two forms of arsenic, he does state that the experiment tends to support the theory that there is interconversion of arsenate to arsenite, which is the more acutely toxic form.

A 2-year feeding study was performed by Byron et al. (1967) on Osborne-Mendel rats and beagle dogs using sodium arsenite and sodium arsenate. For the rat experiment, the sodium arsenite dose levels ranged from 0 to

250 ppm; and for sodium arsenate they ranged from 0 to 400 ppm. The most significant effect from treatment with each arsenical was a marked enlargement of the common bile duct, which was accompanied by a 10-fold thickening of the endothelium. Both agents produced the same effect; the incidence and severity of the enlargement was slightly higher in the highest two arsenate dose groups than the highest two arsenite groups. Dogs treated with arsenate or arsenite experienced weight loss and high mortality, but displayed no histological features remarkably different from controls. The degree of mortality and weight loss was equivalent in the highest and second highest dose groups. In rats and dogs, both forms of arsenic produced the same qualitative toxic effects; the trivalent arsenite was about twice as potent as the pentavalent arsenate.

Savchuck et al. (1960) investigated the differential toxicity (expressed as growth inhibition) of arsenite and arsenate in cultured HeLa cells. Both arsenate and arsenite were found to inhibit the growth of cultured

human liver cells. The levels of arsenic necessary to inhibit growth by 50% were 2.0 ppm of sodium arsenate (pentavalent arsenic) and 0.13 ppm of sodium arsenite (trivalent arsenic). Arsenite, therefore, is approximately 15 times more toxic than arsenate in this in vitro system. Growth inhibition is a definable toxicological end point as is acute toxicity, teratogenicity, and weight loss. Therefore, this study is of value in evaluating similarity of toxic effects.

The studies discussed above demonstrate that both forms of arsenic produce the same qualitative toxic effects, and that the trivalent arsenite is about twice as potent as the pentavalent species. These studies lend support to findings primarily by Ginsburg (1965) and Ginsburg and Lotspeich (1963) that pentavalent arsenic is converted to the trivalent form in vivo.

(3) Equilibrium

Under anaerobic conditions, there is in vitro evidence of an equilbrium between arsenate and arsenite that favors arsenite. The following study by Crecelius (1977a), using wine fermentation (anaerobic process) indicates a four-to-one ratio of arsenite to arsenate regardless of whether arsenite or arsenate were added to a grape juice/yeast mixture. This study demonstrates

that living systems (i.e., yeast) do not affect the valence state of arsenic in an indiscriminant manner. Under a given set of physical and biological conditions the relative proportion of arsenite and arsenate will reach an equilibrium ratio. The arsenic is never all in the form of arsenate nor all in the form of arsenite. If an equilibrium between arsenate and arsenite is attained under anaerobic conditions (i.e., fermentation), it would be logical to assume that under aerobic conditions (the predominant condition in mammals) an equilibrium ratio would also exist. The concept of an equilibrium between arsenate and arsenite suggests that a certain amount of arsenite will be formed whenever a living system is exposed to arsenate. The studies by Ginsburg lend support to this theory.

Crecelius (1977a) studied the metabolic interconversion of inorganic arsenite and arsenate by yeast in the fermentation of grape juice [see Section I.G.(9)]. Trivalent arsenic (arsenite), pentavalent arsenic (arsenate), MAA, and DMAA were added to four equal samples of concord grape juice with wine yeast. The amounts of the four kinds of arsenic were determined after two weeks when the fermentation had stopped. The results showed that the fermentation process reduced approximately 60% of the initial pentavalent arsenic

to the trivalent form, but did not methylate the trivalent arsenic and did not demethylate the MAA. The author also found that, of the arsenic in the final solutions, the final ratio of arsenite to arsenate concentration was about four to one. These results indicate that pentavalent or trivalent arsenic is changed in the fermentation process to yield the same equilibrium ratio, a 4 to 1 ratio of arsenite to arsenate (EPA 1978), regardless of the starting material. Creceluis also found in a survey of 19 domestic table wines, that the arsenite to arsenate ratio averaged about four to one.

It is likely that arsenate will be favored in aerobic conditions that might exist in intact animals. Arsenite

will be favored in anaerobic systems such as wine fermentation. Ferguson and Gavis (1972) have suggested that oxidation and reduction may occur in aerobic and anaerobic natural waters, respectively.

II. REGULATORY HISTORY

Inorganic arsenicals have been the subject of numerous Federal regulatory actions since they were originally registered as pesticides. These actions have been initiated by both the U.S. Department of Agriculture and EPA.

Pesticide Registration (PR) Notice 67-2 issued on August 1, 1967, called attention to "numerous accidents involving children and domestic animals" caused by products containing sodium arsenite and arsenic trioxide. Such products labeled for home use were required to contain no more than 2.0% sodium arsenite nor more than 1.5% arsenic trioxide. In addition, labels of commercial products containing these two compounds were required to warn against storage in or around the home and against allowing domestic animals to graze on treated areas (USDA 1967).

PR Notice 68-3 issued January 10, 1968, classified use of calcium arsenate on droppings under caged layers in poultry houses as a non-food use, and allowed its registration for this use to continue in the absence of finite tolerances (USDA 1968a).

PR Notice 68-8 issued April 24, 1968, classified use of calcium arsenate on tree fruits and use of paris green as a special mosquito larvicide as non-food uses, and allowed registrations for these uses to continue in the absence of finite tolerances (USDA 1968b).

PR Notice 68-19 issued November 29, 1968, required labeling statements for certain economic poisons to protect honeybees and other pollinating insects (USDA 1968c). Arsenical pesticides were listed as "highly toxic" to bees, and labels of such products intended for foliage treatment on agricultural crops, mosquito abatement treatments, and foliage treatment on forest or shade trees were required to bear the warning, "This product is highly toxic to bees exposed to direct treatment or residue on crops."

PR Notice 71-7 issued May 7, 1971, announced that EPA was undertaking an active internal review of arsenicals and other pesticides to determine if any of the presently registered products presented questions of safety substantial enough to initiate the administrative process of cancellation (EPA 1971a).

A series of PR Notices (USDA 1968d; 1968e; 1968f; 1969a; 1969b; 1969c; and 1970a; and EPA 1971b) were issued over several years, extending certain "no residue" and "zero tolerance" registrations of various inorganic arsenicals, based on pending petitions for finite tolerances. Such extensions were granted for use of sodium arsenite on grapes, potatoes, aquatic weeds, beef cattle, goats, and sheep; and for use of lead arsenate on almonds, cabbage, lettuce, potatoes, spinach, turnips, and walnuts.

Another series of PR Notices (USDA 1968g; 1969d; and 1970b; and EPA 1972) were issued to cancel registrations of certain inorganic arsenicals for use on food in the absence of finite tolerances or exemptions. Such cancellations were

issued for the use of arsenic trioxide, calcium arsenate, lead arsenate, copper acetoarsenite, sodium arsenate, and sodium arsenite on livestock and numerous food and feed crops.

III. EXPOSURE ANALYSIS

In subsequent portions of this document the Working Group will discuss the potential adverse effects of inorganic arsenicals. The Agency's decision whether to permit the registration or continued registration of these pesticides will be based, to a large extent, upon anticipated human exposure resulting from their use.

This preliminary exposure analysis discusses representative exposure situations as a basis for comment by interested parties. Before the Agency makes any regulatory decision concerning inorganic arsenical pesticides, a complete risk and benefit analysis will be performed, and the benefits of use of these pesticides will be weighed against their potential risks.

The Working Group emphasizes that a safe level for carcinogenic or mutagenic substances has never been established. Therefore, any level of a carcinogenic or mutagenic substance may cause an adverse effect and the Agency does not consider exposure when determining whether to issue an RPAR based on carcinogenic or mutagenic

effects. Exposure is considered, however, when determining whether or not to issue an RPAR based on other chronic effects such as teratogenicity (birth defects). This analysis will address estimated exposure from arsenical pesticides to applicators and the general public.

Arsenical pesticides differ from synthetic organic pesticides in that arsenic is a natural element and does not degrade to less toxic constituents; therefore, the arsenic added to the environment through pesticide application will either remain as residues in the soil where applied, or be removed from the site by plant uptake, erosion, or volatilization. Since arsenic undergoes biological and chemical transformations, the arsenic from pesticides becomes indistinguishable from that occurring naturally.

The Working Group will examine representative exposure situations for each inorganic arsenic compound (see Table 8).

Table 8. Representative Exposure Situations

Agricultural Exposure (including Home and Garden)
1. Applicator exposure resulting from the use of arsenical pesticides on food crops, weeds, etc.; and
2. Exposure resulting from the consumption of food or feed crops treated with arsenical pesticides;
Exposure Resulting From the Treatment of Wood With Arsenical Wood Preservatives and Subsequent Use of Treated Wood
1. Large-scale industrial situations;
2. Intermittent application to wood during on site construction;
3. Exposure from the handling, sawing, and use of treated wood products; and
4. Exposure to individuals living or working in dwellings containing arsenic-treated wood.

When there is the potential for teratogenic effects, the Working Group must determine whether an ample margin of safety exists between the level of arsenic which produces teratogenic effects and the level to which humans can reasonably be anticipated to be exposed. Teratogenic effects occur only when the woman is exposed to the chemical during pregnancy. For this reason the Working Group will base its calculations on a 60-kg female applicator of child-bearing age.

A. Agricultural Exposure (Including Home and Garden)

The following inorganic arsenical pesticides are registered with the Environmental Protection Agency: arsenic acid, ammonium arsenite, arsenic trioxide, arsenic pentoxide, calcium arsenate, copper acetoarsenite, lead arsenate, sodium arsenite, sodium arsenate, and sodium pyroarsenate. Each of these pesticides, with the exception of sodium pyroarsenate, ammonium arsenite, and arsenic pentoxide, have agricultural or home uses. The Working Group will evaluate the oral, dermal, and inhalation exposure to those arsenical pesticides registered for agricultural or home use. Sodium pyroarsenate, ammonium arsenite, and arsenic pentoxide are used in the preservation of wood and will be discussed in subsequent portions of this report.

Because of the lack of data concerning exposure, the Working Group will make the following assumptions:

1. For analysis of oral exposure, the Working Group assumes that arsenic residues will be present at currently authorized tolerance levels. The Working Group realizes that residues found in or on raw agricultural commodities are typically lower than tolerance levels; however, because residues could lawfully be present at tolerance levels it is prudent to assume tolerance levels for regulatory pruposes. The arsenic contribution of each food will be calculated based on the average daily intake of the commodity as reported by Lehman (1962). Iwataki and Horiuchi (1959) sampled feces, urine, and blood of a normal (Japanese) population for arsenic. More than half of the arsenic detected (form not indicated) was found in the blood and urine. It is assumed, therefore, that 75% of all arsenic ingested will be absorbed;

2. For analysis of dermal exposure, it is assumed that the applicator will be contaminated with up to one pint of liquid (diluted for application per label instructions) and up to 10 grams of dust (in the case of dust formulations). It is further assumed that 10% of all arsenic in contact with the skin will be absorbed. Oehme (1972) states that inorganic arsenic is absorbed significantly through the intact skin; and

3. For analysis of inhalation exposure, it is assumed the applicator's breathing rate is 1.8 m^3/hr (active rate) and that all material inhaled will be absorbed. The Working Group also assumes that the applicator would spray for approximately seven hours per day.

(1) Sodium Arsenite

The Working Group has selected a product for analysis which contains 42.5% sodium arsenite as formulated (equivalent to 1.05 kg arsenic/gal). When diluted according to label directions, the solution contains 3.28 grams of arsenic per pint.

(a) Oral Exposure

In order to calculate the theoretical quantity of arsenic in the average diet, the Working Group will use the following formula, A x B x C x D / E in which:

A = the theoretical average daily intake of sodium arsenite in mg/kg, expressed as As_2O_3;

B = 1.5 kg, the average daily food intake (Lehman 1962);

C = 0.76, a correction factor for conversion from milligrams of the arsenic compound (As_2O_3) to total arsenic (the molecular weight of arsenic divided by the molecular weight of the compound);

D = 0.75% absorption factor; and

E = 60 kg. (Exposure is normally expressed in terms of milligrams of chemical per kilogram of body weight. Because teratogenic effects are of concern with inorganic arsenic all calculations of exposure will be based on exposure to a 60-kg woman.)

Therefore, for oral exposure to sodium arsenite (see Table 9), A x B x C x D / E = 0.051 mg/kg X 1.5 kg X 0.76 X 0.75 / 60 kg = 0.0007 mg/kg arsenic.

Table 9. Arsenic in Daily Diet Resulting From the Use of Sodium Arsenite

Food Item	Food Factor (percent of total diet)	Tolerance (mg/kg) [40 CFR 180.335]	Contribution to daily intake (mg/kg a/)
Beef b/	4.60	0.7	0.0322
Horse b/	data not available	0.7	---
Edible Offals c/	0.69	2.7	0.01863
Grapes	0.32	0.05	0.00016
Total	5.61		0.05099 mg/kg

a/ Expressed as As_2O_3.
b/ Meat, fat, and meat by-products.
c/ Liver and kidney.

(b) Dermal Exposure

Assuming a one-pint exposure during mixing, handling, and application, an applicator would receive 3.28 grams X 0.1 (absorption) = 0.328 gm, or 328 mg; 328 mg / 60 kg = 5.47 mg arsenic/kg of body weight from dermal exposure to sodium arsenite.

(c) Inhalation Exposure

Using an estimate of 100 ug/m^3 [based on the concentration of arsenic in sodium arsenite and on extrapolation of data from Pinto et al. (1976)] for the concentration of metallic arsenic and the assumptions stated above, the inhalation exposure for a 7-hour day would be approximately 100 ug/m^3 X 1.8 m^3/hr X 7 hrs = 1,260 ug or 1.260 mg arsenic; 1.260 mg / 60 kg = 0.021 mg arsenic/kg of body weight from inhalation exposure to sodium arsenite.

Total theoretical exposure from sodium arsenite equals:

0.0007 mg/kg (through food)
5.47 mg/kg (due to dermal exposure)
0.021 mg/kg (due to inhalation exposure)
5.5 mg/kg

(2) Arsenic Acid

The Working Group has selected a representative arsenic acid product for analysis which contains 17.5% arsenic acid as formulated (equivalent to 1.5 pounds arsenic per gal). When diluted according to label directions, the solution contains 1.625 grams of arsenic per pint.

Table 10. Arsenic in Daily Diet Resulting From the Use of Arsenic Acid

Food Item	Food Factor (percent of total diet)	Tolerance (mg/kg) [40 CFR 180.180]	Contribution to daily intake (mg/kg[a/])
cottonseed	0.6[b/]	4	0.0316
Total	0.6		0.0316 mg/kg

a/ Expressed As_2O_3.

b/ Cottonseed is used in edible oils, margarine, etc. This food factor is an estimate and is not found in Lehman (1962).

(a) Oral Exposure

The theoretical quantity of arsenic in the daily diet due to arsenic acid (see Table 10) equals A x B x C x D / E (see oral exposure analysis for sodium arsenite), or 0.0316 mg/kg X 1.5 kg X 0.76 X 0.75 / 60 kg = 0.0005 mg/kg arsenic.

(b) Dermal Exposure

Assuming a one-pint exposure, an applicator would receive 1.625 grams x 0.1 (absorption) = 0.1625 gm, or 162.5 mg; 162.5 mg / 60 kg = 2.7 mg arsenic/kg of body weight from dermal exposure to arsenic acid.

(c) Inhalation Exposure

Using the estimate of 100 ug/m^3 [based on extrapolation of data from Pinto et al. (1976)] for the concentration of arsenic and the assumptions stated above, the inhalation exposure for a 7-hour day would be 100 ug/m^3 X 1.8 m^3/hr X 7 hr = 1260 ug, or 1.260 mg arsenic; 1.260 mg / 60 kg = 0.021 mg/kg arsenic from inhalation exposure to arsenic acid.

Total theoretical exposure from arsenic acid equals:

0.0005 mg/kg (through food)

2.7 mg/kg (dermal exposure)

0.021 mg/kg (inhalation exposure)

2.72 mg/kg

In addition to the exposure situations discussed above, the Working Group is also interested in arsenic exposure resulting from ginning of cotton treated with arsenic acid (as a desiccant). As a representative exposure situation, the Working Group will assess exposure to individuals living or working close to a cotton gin processing cotton treated with arsenic acid. Suta (1978) provided data showing the estimated arsenic in air concentrations at various distances from cotton gins. Arsenic concentrations were estimated to range from 0.003 ug/m^3 to 29 ug/m^3 for

various cotton gins at various distances. Assuming all arsenic in air to be respirable and the concentration to be 29 ug/m^3, inhalation exposure for a 60-kg woman would be: 1.0 m^3/hr (breathing rate of a resting person) X 29 ug/m^3 (concentration of arsenic in air) / 60 kg = 0.48 ug/kg per hour. Assuming exposure to continue 24 hours per day, exposure would be 11.60 ug/kg, or 0.0116 mg/kg per day. These exposure estimates are for individuals living near cotton gins. Exposure to workers inside the gin would be significantly higher.

(3) Sodium Arsenate

(a) Oral Exposure

Although sodium arsenate has established tolerance levels for use on grapes, no sodium arsenate products are registered for such use; therefore, arsenic residues will not be present due to the use of sodium arsenate.

(b) Dermal Exposure

The Working Group has selected a representative sodium arsenate product containing 3% sodium arsenate as formulated (1.3% as arsenic) which is used in combination with sugar as an ant bait. Dermal exposure to the ant bait would generally be low; however, the directions call for saturating cotton with the 1.3% arsenic solution. In this case, the applicator could

reasonably be expected to contact the wet cotton and be dermally exposed to as much as 30 gm (2 tablespoons) of solution. By calculation this would be 30 gm X 0.013 (% arsenic) = 0.39 gm, or 390 mg of arsenic metal. If 10% is absorbed by a 60-kg woman then the theoretical dermal dose would be: 390 mg X 0.10 = 39 mg; 39 mg / 60 kg = 0.65 mg/kg arsenic from dermal exposure to sodium arsenate.

(c) Inhalation Exposure

Since the product discussed above is used as a very dilute liquid, not a spray, inhalation exposure to sodium arsenate would not be significant.

Total theoretical exposure to sodium arsenate, therefore, would be 0.65 mg/kg (resulting from dermal exposure).

(4) Copper Acetoarsenite

The Working Group has selected a representative copper acetoarsenite (paris green) product which is used as a mosquito larvicide and contains 3.56% arsenic as the metal. The product is ready to use and does not require dilution. If applied aerially, minimal exposure to the applicator is expected. If applied by hand duster, dermal and inhalation exposure may result.

(a) Oral Exposure

Copper acetoarsenite has no registered food uses.

(b) Dermal Exposure

Wolfe et al. (1967) investigated dermal and inhalation exposure resulting from the use of insecticide dusts. Applicators using a power duster to apply 4% malathion dust to beans were exposed to 23 mg/hr dermally and 0.73 mg/hr by inhalation. Assuming the conditions and the equipment were similar, exposure from the use of copper acetoarsenite dust (containing 3.56% arsenic as the metal) can be calculated by incorporating a factor to correct for the difference between the concentration of pesticide in Wolfe's study (4%) and the arsenic concentration in copper acetoarsenite:

$$\text{Correction Factor } \frac{3.56\% \text{ (concentration of arsenic)}}{4\% \text{ (concentration of malathion)}} = 0.89.$$

Therefore, dermal exposure would be equal to:

23 mg/hr x 0.89 (correction factor) x 7-hours (length of work day) x 0.1 (absorption) / 60 kg = 0.24 mg/kg.

(c) Inhalation Exposure

Using Wolfe's information as discussed above, the inhalation exposure for a 7-hour work day is estimated to be:

0.73 mg/hr x 0.89 (correction factor) x 7 hours / 60 kg = 0.08 mg/kg.

Total theoretical exposure for copper acetoarsenite equals:

0.24 mg/kg (due to dermal exposure)
0.08 mg/kg (due to inhalation exposure)
0.32 mg/kg

(5) Calcium Arsenate

For the dermal and inhalation exposure analyses, the Working Group has selected a dust representative product containing 5% calcium arsenate (equivalent to 22.73 gm calcium arsenate per pound).

(a) Oral Exposure

Table 11. Arsenic in Daily Diet Resulting From the Use of Calcium Arsenate

Food Item	Food Factor (percent of total diet)	Tolerance (mg/kg) [40 CFR 180.192]	Contribution to daily intake (mg/kg a/)
Asparagus	0.23	3.5	0.0081
Beans	1.24	3.5	0.0434
Berries b/	0.06	3.5	0.0021
Blackberries c/	0.03	3.5	0.0011
Broccoli	0.19	3.5	0.0067
Brussels Sprouts	0.16	3.5	0.0056
Cabbage d/	0.49	3.5	0.0172
Carrots	0.55	3.5	0.0193
Cauliflower	0.43	3.5	0.0151
Celery	0.76	3.5	0.0256
Corn	1.19	3.5	0.0417
Cucumbers	0.73	3.5	0.0256
Egg plant	0.32	3.5	0.0112
Kale e/	0.14	3.5	0.0049
Melons	1.98	3.5	0.0693
Peppers	0.25	3.5	0.0088
Raspberries c/	0.04	3.5	0.0014
Rutabagas	data not available		
Spinach	0.29	3.5	0.0102
Squash f/	0.19	3.5	0.0067
Tomatoes	3.34	3.5	0.1169
Turnips	data not available		
Total	12.16		0.4409

a/ Expressed as As_2O_3.
b/ Includes blueberries, boysenberries, and huckleberries.
c/ Includes dewberries, loganberries, and youngberries.
d/ Includes kohlrabi.
e/ Includes pumpkins.

The theoretical quantity of arsenic in the daily diet (see Table 11) due to calcium arsenate equals A X B X C X D / E (see the oral exposure analysis for sodium arsenite) = 0.4409 mg/kg X 1.5 kg X 0.76 X 0.75 / 60 kg = 0.006 mg/kg.

(b) Dermal Exposure

Using Wolfe's observations as discussed in Section III. (4)(C) (Copper Acetoarsenite), dermal exposure would be equal to 23 mg/hr X 1.2 (correction factor for calcium arsenate) x 0.1 (absorption) X 7 hours X 0.38 (percentage of arsenic in calcium arsenate) / 60 kg = 0.12 mg/kg.

(c) Inhalation Exposure

Using Wolfe's information concerning inhalation, exposure would equal 0.73 mg/hr X 1.2 (correction factor) X 7 hours X 0.38 (percent arsenic) / 60 = 0.04 mg/kg.

Total theoretical exposure from calcium arsenate equals:

0.006 mg/kg (through food)
0.12 mg/kg (through dermal exposure)
0.04 mg/kg (due to inhalation exposure)
0.17 mg/kg

(6) Arsenic Trioxide: Oral, Dermal, and Inhalation Exposure

The Working Group has selected a representative arsenic trioxide product which contains 1.5% As_2O_3 and is used for control of mice, ants, and roaches. This bait is used full strength for rodents and diluted with meal for insects. The actual metallic arsenic concentration is 1.136%.

Due to the nature of the product, it is doubtful that oral or inhalation exposure would be significant. Carelessness and ordinary transfer problems could result in a dermal exposure of 2 tablespoons of solution on the hands. Two tablespoons contains approximately 30 gm of formulated product. Dermal exposure would then equal: 30 gms X 0.10 (10% absorption) X 0.0136 (% arsenic) = 0.0408 gm, or 40.8 mg arsenic; 40.8 mg / 60 kg = 0.68 mg/kg arsenic from dermal exposure to arsenic trioxide.

(7) Lead Arsenate

The Working Group has selected a representative lead arsenate product which contains 80% lead arsenate as formulated (equivalent to 0.03 pounds of lead arsenate/gallon) and when diluted according to label directions will contain 0.375 grams arsenic per pint.

Table 12. Arsenic in Daily Diet Resulting From the Use of Lead Arsenate

Food Item	Food Factor (percent of total diet)	Tolerance (mg/kg) [40 CFR 180.194]	Contribution to daily intake (mg/kg[a/])
Apples	1.81	7[b/]	0.1267
Apricots	0.12	7	0.0084
Asparagus	0.23	7	0.0161
Avocados	0.03	7	0.0021
Berries[c/]	0.06	7	0.0042
Blackberries[d/]	0.03	7	0.0021
Celery	0.76	7	0.0532
Cherries	0.17	7	0.0119
Cranberries	0.10	7	0.007
Currants[e/]	0.12	7	0.0084
Eggplant	0.32	7	0.0224
Grapes	0.32	7	0.0224
Mangoes	data not available		
Nectarines	0.06	7	0.0042
Peaches	1.03	7	0.0721
Pears	0.32	7	0.0224
Peppers	0.25	7	0.0175
Plums (fresh)	0.23	7	0.0161
Quinces	data not available		
Raspberries[d/]	0.04	7	0.0028
Strawberries	0.19	7	0.0133
Tomatoes	3.34	7	0.2338
Citrus Fruits	4.00	7	0.04
total	13.53	1[f/]	0.7071

a/ Expressed as combined lead.
b/ Equivalent to 3.5 ppm As_2O_3.
c/ Includes huckleberries, boysenberries, and blueberries.
d/ Includes dewberries, loganberries, and youngberries.
e/ Includes gooseberries.
f/ Equivalent to 0.5 ppm As_2O_3.

(a) Oral Exposure

The theoretical quantity of arsenic in the daily diet due to lead arsenate (see Table 12) equals A X B X C X D / E (see oral exposure analysis for sodium arsenite) = 0.354 mg/kg (equivalent to 0.707 combined lead) X 1.5 kg X 0.76 X 0.75 / 60 = 0.005 mg/kg arsenic.

(b) Dermal Exposure

Assuming a one-pint exposure, an applicator would receive 0.375 gm X 0.1 (10% absorption) = 0.0375 gm, or 37.5 mg; 37.5 mg / 60 kg = 0.63 mg/kg arsenic from dermal exposure to lead arsenate.

(c) Inhalation Exposure

Using the estimate of 100 ug/m^3 [based on extrapolation of data from Pinto et al. (1976)] for the concentration of arsenic, and the assumptions stated above, the inhalation exposure could be 100 ug/m^3 X 1.8 m^3/hr X 7 hr = 1,260 ug, or 1.260 mg arsenic; 1.260 mg / 60 kg = 0.021 mg/kg arsenic from inhalation exposure to lead arsenate.

Total theoretical exposure from lead arsenate equals:

0.006 mg/kg (through food)
0.63 mg/kg (dermal exposure)
0.021 mg/kg (inhalation exposure)
0.66 mg/kg

B. Exposure From Treatment of Wood With Arsenical Wood Preservatives and Subsequent Use of Such Products

Arsenical salts are used to protect a variety of wood products against various pests. Arsenic treated wood is used in the construction of homes, in utility poles, fence posts, marine piers, pilings, etc. The majority of wood is treated in closed cylinders under pressure, although there are instances where the preservatives are painted on wood or wood is soaked in solutions containing arsenic.

There are three basic arsenical wood preservatives of commercial importance. These are Chromated Copper Arsenate (CCA), Ammonical Copper Arsenate (ACA), and Fluor Chrome Arsenate Phenol (FCAP). These three wood preservatives are various mixtures made, in part, from either ammonium arsenite, arsenic trioxide (both trivalent arsenicals), arsenic acid, arsenic pentoxide, sodium arsenate, or sodium pyroarsenate (all pentavalent arsenicals). During the formulation process all trivalent arsenic is oxidized to pentavalent arsenic (AWPI 1977).

In the past, arsenical wood preservatives have been referred to under many different names. The various names and relationships of chemical constituants are cross-referenced in Table 13. The percentages listed indicate

the relative proportion of active ingredients in the formulated products. The percentages listed are on the oxide (solids) basis. Table 14 lists typical arsenical formulations available.

Table 13.[a/]

CHROMATED COPPER ARSENATE (CCA, Cr-Cu-As, CKA)

Type	Chromium as CrO_3	Copper as CuO	Arsenic as As_2O_3	Common Name
A	65.5%	18.1%	16.4%	CCA-A CCA-Type I Ascu Greensalt-K Erdalith
B	35.3%	19.6%	45.1%	CCB-B CCA-Type II Boliden Salt K-33 Lahontuho K-33
C	47.5%	18.5%	34.0%	CCA-C Tenalith C Celcure AP Chrom- Ar-Cu Tancas C Wolman CCA K-33-C

AMMONICAL COPPER ARSENATE (ACA, Chemonite)

Copper as CuO	Arsenic as As_2O_3	Ammonia NH_3	Common Name
49.8%	50.2%	weight of NH_3 1.5-2.0 times weight of CuO	CCA Chemonite

FLUOR CHROME ARSENATE PHENOL

Fluoride as F	Chromium as CrO_3	Arsenic As_2O_3	Dinitrophenol of Sodium Pentachlorophenate	Common Name
22%	37%	25%	16%	FCAP Tanalith U CFA Osmosalts

a/ AWPI (1977)

Table 14. Representative Formulations of Pesticide Products Used in the Pressure Treatment of Wood

CCA-Type A		CCA-Type B	
Potassium Dichromate	53%	Arsenic Pentoxide*	34%
Copper Sulfate	32%	Copper Oxide	14.5%
Arsenic Pentoxide*	10%	Chromic Acid	25.6%
Inerts	5%	Inerts	25%
*(Arsenic as the metal	5.64%)	*(Arsenic as the metal	22.17%)

CCA-Type C	
Arsenic Pentoxide*	26.9%
Sodium Dichromate	42.6%
Copper Sulfate	25.5%
Inert	5.9%
*(Arsenic as the metal	14.09%)

Ammonical Copper Arsenate (ACA)+	
Arsenic Trioxide*	37%
Copper Metallic	36%
*(Arsenic as the metal	28.12%)

+This is not sold as a formulated product. These chemicals are used to produce an intermediate which is diluted on site to prepare the dilute treating solution. There are no formulated products for ACA preservatives intended for industrial use.

Fluor Chrome Arsenate Phenol	
Sodium Chromate	34.2%
Sodium Floride	27.5%
Disodium Arsenate*	23.8%
Sodium Pentachlorophenate	7.8%
Sodium Salts of other chlorophenol & related compounds	1.7%
Inert	5.0%
*(Arsenic as the metal	9.59%)

The following is an example of activities associated with industrial wood treatment situations although individual treatment facilities may vary their procedure. In the industrial situation arsenical formulations (concentrates)

are delivered as dusts, liquid concentrates, or pastes. The concentrates are dumped or otherwise metered and mixed with water to form dilute treating solutions of 0.75 to 6.0%, depending on the intended end-use of the treated wood.

Lumber to be treated is stickered (stacked so as to provide spaces for fluid circulation) onto a tram either by machine or hand. The loaded tram is moved (e.g., by rail) into the treatment cylinder. The cylinder door is closed and air in the cylinder evacuated. The cylinder is then filled with treating solution from storage tanks. Pressure is applied to achieve maximum impregnation, a process which may require up to eight hours. The cylinder is emptied by air pressure upon completion of the treatment cycle.

Following removal of the treating solution, the cylinder door is unbolted and pulled open manually. The charged lumber is pulled from the cylinder on its tram to a concrete pad where drippings are collected for reuse. The treated product is removed from the tram by forklift truck for air drying in the yard or kiln. The treated lumber is unstickered by laborers who also stamp on the trademark and stack the lumber for shipment.

Exposure from treatment of wood or its subsequent use can be divided as follows: large scale industrial situations; intermittent application to wood during on-site construction;

exposure from handling, sawing, fabrication, etc., of treated wood products; and exposure to individuals living or working in dwellings containing arsenic-treated wood.

(1) Large Scale Industrial Situations

The regulation of industrial situations to insure worker safety is normally the responsibility of the Occupational Safety and Health Administration. However, Section 4(b)(1) of the Occupational Safety and Health Act (29 U.S.C. S653(b) (1)) provides that "[n]othing in this Act shall apply to working conditions of employees with respect to which other Federal Agencies...exercise statutory authority to prescribe or embrace standards or restrictions affecting occupational safety or health." Since the industrial treatment of wood with arsenic preservatives involves the application of a pesticide, EPA has assumed regulatory responsibility under FIFRA. EPA regulation of arsenic in this use pattern has the effect of pre-empting OSHA. Exposure to inorganic arsenic in the industrial setting could arise through the inhalation and/or dermal routes.

(a) Inhalation Exposure

Several studies have been conducted to determine the amount of arsenic in air in or around treatment plants during the treatment and handling of treated wood (Table 15). Most of this data was presented by Dr. Robert Arsenault during OSHA public hearings in April 1975 on the proposed exposure standard for inorganic arsenic. Dr. Arsenault noted that "a large amount of the data was obtained during damp winter conditions so that the numbers (concentration of arsenic in air) shown are conservative estimates. In dry summer weather, the airborne arsenic from handling lumber, such as in air seasoning yards and during unstickering, would be much higher" (Arsenault 1975).

Table 15. Arsenic in Air During Treatment and Handling of Wood[a/]

Chemical	Activity	Arsenic Concentration in Air (mg/m^3)	Comment
ACA	changing retort charge	0.012	
ACA	boring of treated wood to assay for arsenic	0.001	
ACA	mixing treatment charges, moving barrels containing arsenic to door of mixer, dump chemical, scrape chute	0.020 0.025	light rain
ACA	hand stickering of treated wood	0.006 0.004 0.004	light rain
ACA	loading treated poles on flat car	0	
ACA	wiring and strapping treated lumber on flat car	0.007 0.002	
ACA	loading and stickering of treated poles in kiln driers	0 0.002	light rain
ACA	machine stickering of treated plywood	0.003 0.0034 0.002	
ACA	hand unstickering of treated lumber	0.020 0.007 0.055	
ACA	unloading of kiln drier; moving material from kiln to shed	0.001 0.005 0.007	
ACA	five feet down wind from stack of air drying poles	0.002 0.003 0.004	
CCA	exhaust of drying kiln fully charged with treated lumber	0.0042	sample was taken at the exhaust of kiln by inserting probe directly into the gas stream as it was exhausted

a/ Data from Arsenault (1975).

Table 15 (continued)

Chemical	Activity	Arsenic Concentration in Air (mg/m^3)	Comment
CCA	surface area of a bundle of treated lumber just out of the retort	0.0014	
CCA	treating wood	0.008	
CCA	stacking dry treated lumber	0.002	
CCA-B	treated lumber stored outside	0.001	
CCA-B	mixing/treating area	0.001	no mixing underway no forced ventilation
CCA-B	changing the charge of treatment tank	0.03	
CCA-C	mixing preservative	0.05	concentrate mixed in open tanks
CCA-C	in lumber yard of treatment plant	0.01	
CCA-C	stacking dry treated plywood	0.01	
CCA-C	stacking dry treated plywood	0.22	
CCA-C	handling treated lumber in lumber yard	0.0018	
CCA-C	stickering and unstickering of treated wood	0.007	
CCA-C	locomotive operator and helper moving treated wood from treatment site site	0.0001	
CCA-C	taking test sample (boring) of treated wood at treatment site	0.0001	

Table 15 (continued)

Chemical	Activity	Arsenic Concentration in Air (mg/m^3)	Comment
CCA-C	treater loading wood	0.0001	
CCA-C	mixing preservative from dry powder concentrate	0.77 (8 hour TWA for 2 mixes 0.016)	one or two mixes/ day; each mix takes 8.8 to 9 min, operator handles dry wood the remainder of the day, opera- used respirator, gloves, and apron

The highest arsenic-in-air values (0.77 mg/m^3) were detected when workers mixed preservatives from dry preservative concentrates. This activity, however, does not continue for extended periods of time. As noted by the author, one or two mixes are prepared each working day with each mixing operation lasting 8.8 to 9.0 minutes. The 8-hour time-weighted average (TWA) for this activity would be approximately 0.016 mg/m^3.

The next highest arsenic-in-air value (0.22 mg/m^3) was detected when workers stacked dry, treated plywood. In order to calculate the theoretical inhalation exposure resulting from this activity, the Working Group assumes that the workers' breathing rate is 1.8 m^3/hr; that all inhaled arsenic will be respirable; that all arsenic reaching the lung will be absorbed; and that the worker will spend the entire working day (8 hours) in the contaminated areas.

Therefore, 0.22 mg/m^3 X 1.8 m^3/hr (breathing rate) = 0.396 mg arsenic/hr; 0.396 mg/hr X 8 hours per day = 3.168 mg arsenic inhaled per day. If we assume the worker to be a 60-kg woman, then exposure would be 3.168 mg / 60 kg = 0.05 mg/kg per day.

The Pacific Biomedical Research Center, University of Hawaii (PBRC Undated), conducted urine analysis studies of workers involved in the industrial application of arsenical (CCA) preservatives. The purpose of the study was to estimate arsenic exposure by monitoring arsenic concentration in urine. The study population was divided into two groups "reference" and "exposed." The reference group corresponded to workers employed in the wood treating industry whose contact with CCA preservatives was "incidental or ambiguous such as clerks, salesman, chemists, plant superintendents, etc." The author concluded there was not a significant difference in residue levels between the "exposed" and "reference" groups. The author did not, however, compare the arsenic concentration of the "reference" group to that of the local population not associated with the preservative (Letter 1978). Mean arsenic values of individuals in the study were as follows: "Exposed" (42 individuals), 0.074 ppm; "Reference" (12 individuals), 0.070 ppm.

The author indicated there may have been an incidental exposure to arsenic by the "reference group." The author also indicated that the plant was almost entirely in the open (Letter 1978).

(b) Dermal Exposure

Little data is available with which to assess potential dermal exposure to arsenic in the industrial situation. If we assume, however, that the actual treatment of wood occurs in closed cylinders under pressure and that there is no exposure to the worker during treatment, then dermal exposure will be limited either to handling preservative concentrates and dilute treating solutions, or to handling wet treated wood after treatment. The following analysis assesses the potential dermal exposure resulting from the use of dust and liquid concentrate formulations.

(i) Handling Preservative Concentrates and Dilute Treating Solutions

a. Dust Formulations

Fluor Chrome Arsenate Phenol (FCAP) preservative formulations are shipped as dusts. The Working Group assumes that workers involved in dumping dust concentrates to prepare dilute treating solutions will each be contaminated with up to 10 grams of dust concentrate. Representative FCAP formulations contain 9.59% arsenic as the metal. The

Working Group assumes that 10% of all dust coming in contact with the worker will be absorbed. Therefore, 10 gm (exposure) X 0.0959 (% concentration of arsenic as metallic) = 0.959 gm, or 959 mg; 959 mg X 0.1 (% absorbed) = 95.9 mg. If we assume the worker to be a 60-kg woman, exposure would be 95.9 mg / 60 kg = 1.6 mg/kg arsenic exposure to FCAP concentrate wood preservatives.

b. Liquid Formulations

CCA preservatives are often shipped as liquid concentrates. The Working Group assumes workers involved in preparing dilute CCA solutions could be contaminated with up to 20 ml of liquid concentrate (enough to wet both hands). Representative CCA formulations contain 27.17% arsenic as metallic. The Working Group assumes 10% of all arsenic coming in contact with the worker will be absorbed. Therefore, 20 ml (20 gms) X 0.27 (% arsenic as metallic) = 5.4 gm arsenic reaching the body; 5.4 X 0.1 (% absorption) = 0.54 gm, or 540 mg. If we assume the worker to be a 60-kg woman, exposure would be 540 mg / 60 kg = 9.0 mg/kg arsenic resulting from exposure to CCA concentrate wood preservatives.

Klemmer et al. (1975) studied the amount of arsenic in house dust of persons employed in the wood preserving industry and persons living in homes containing arsenic treated wood. One member of the study group was actually

involved with handling CCA formulated products (Memo 1978b). This home did not contain treated wood and yet contained 1,080 ppm arsenic in house dust, while homes whose residents did not work with arsenic and did not contain treated wood showed levels of 1.1 to 9.1 ppm arsenic in dust. The Working Group assumes the 1,080 ppm arsenic in dust resulted from occupational exposure to CCA wood preservatives and documents the significant nature of worker exposure to arsenic resulting from industrial use of arsenical wood preservatives.

(ii) Handling Wet Treated Wood After Treatment

The Working Group assumes workers in plants using arsenic in the treatment of wood will handle wet, treated wood without gloves or specialized clothing (Memo 1978b) and that the concentration of arsenic on the surface of the wood will be that of the treating solution. If gloves are worn, however, it is assumed these gloves would be made of cloth, would become saturated with treating solution, and would provide no protection against dermal exposure. Treating solutions vary from 0.75 to 6% active ingredient depending on the intended end use of the treated wood (AWPI 1977). Table 14 lists representative preservative formulations. Assuming that the treating solution used will contain 6% active ingredient, the amount of arsenic as metallic in representative dilute treating solutions will be 1.33% for CCA, 1.69% for ACA, and 0.57% for FCAP.

Assuming that the treated wood removed from the treatment cylinders will be dripping wet, that the worker will be contaminated with up to 20 ml (enough to wet both hands) of dilute treating solution, and that 10% of this arsenic-contaminated solution will be absorbed, and using the figure of 1.69% metallic arsenic in dilute Ammonical Copper Arsenate preservatives as an example, the dermal exposure resulting from handling wet treated wood will be as follows: 20 ml (20 gm) X 0.017 (% arsenic as metallic) = 0.34 gm, or 340 mg arsenic reaching the body; 340 mg X 0.1 (% absorption) = 34 mg. If we assume the worker to be a 60-kg woman, the dermal exposure will be 33.8 mg / 60 kg = 0.57 mg/kg resulting from exposure to wet treated wood.

(2) Intermittent Application to Wood During On-Site Construction

Two wood preservative products (containing 7.7% ammonium arsenite, equivalent to 3.26% arsenic) are available for use during the construction of structures built with pressure-treated wood. The products are intended for use on freshly cut ends of previously treated lumber. The directions instruct the applicator (i.e., carpenter) to apply several coats of preservative with a brush or rag mop and to wash

off the solution to avoid staining the hands. Because these products are applied as a viscous liquid, and not as a spray, inhalation exposure to ammonium arsenite would be minimal.

Assuming 20 ml (enough to wet both hands) of solution will reach the skin and or clothing and that 10% will be absorbed before the skin is washed, the applicator could receive 20 ml (20 gm) X 0.033 (% concentration of arsenic) X 0.1 (% absorption) = 0.066 gm, or 66 mg arsenic.

Assuming the applicator to be a 60-kg woman, the applicator would receive 66 mg / 60 kg = 1.1 mg/kg arsenic from dermal exposure to ammonium arsenite.

(3) Exposure from Handling, Sawing, Fabrication, etc., of Treated Wood Proudcts

Exposure to inorganic arsenic under these conditons would arise through the inhalation and/or dermal routes.

(a) Inhalation Exposure

Several studies have been conducted to determine the amount of arsenic in air during cutting and building operations involving treated wood. Table 16 indicates the results from these studies.

The highest value recorded was 0.7 mg/m^3 during the cutting of ACA-treated plywood using a router. The author commented that these results were obtained after only 5 minutes of cutting. In order to conduct this analysis the following assumptions are made:

1. The worker will be involved in sawing related activities for 8 hours a day;
2. The air will not become saturated with arsenic particles in only 5 minutes. The arsenic in air level will be at least double the five minute value, or 1.4 mg/m^3, if this activity continued throughout the working day;
3. The workers breathing rate will be 1.8 m^3/hr;
4. 25% of the arsenic containing dust will be respirable;
5. All arsenic reaching the lung will be absorbed;
6. All of the non-respirable arsenic (not absorbed through lung tissue but present in respiratory system) containing dust will enter the gastro-intestinal tract;
7. Of the 75% non-respirable dust, 75% will be absorbed through the GI tract; and
8. The worker will spend the entire working day in the contaminated area.

Table 16. Arsenic Concentrations in Air During Cutting and Building Operations[a/]

Chemical	Activity	Arsenic Concentration in Air mg/m^3)	Comments
ACA	sawing treated wood indoors (10" table saw)	0.24 0.048 0.042 0.062	results obtained during the rainy season and would be much higher in drier months
ACA	shaving the ends of treated poles	0.019 0.123 0.249	
ACA	cutting treated plywood with a router, plywood treated with 0.6 pcf	0.07-0.70	after cutting 5 min
ACA	cutting plywood with skill saw, plywood treated with 0.6 pcf	0.36	after cutting through feet of board (author notes the respirable fraction was 5.3%, or 0.019 mg/M^3)
ACA	handling and hammering treated wood	0.01-0.033 (TWA) 0.01-0.024)	author notes that these values include some cutting opperations
CCA-B	sawing treated studs	0.038	no forced ventilation in building
CCA-B	Construction of foundation panels using treated wood (assembly area)	0.012	no forced ventilation in building
CCA-B	during assembly of treated wood foundations	0.012	no forced ventilation in building
CCA-C	cutting plywood containing 0.6 pcf using a 6" fine-toothed plywood finish blade	0.071 0.097 0.015	this blade produces less dust per cut than a general purpose blade; data collected indoors, no external ventilation

a/ Data from Arsenault (1975).

Therefore 1.4 mg/m^3 X 1.8 m^3/hr (breathing rate) = 2.52 mg/hr; 2.52 mg/hr X 8 hours/day = 20.16 mg/day; 20.16 mg X 0.25 (% respirable) = 5.04 mg reaching lungs; 20.16 mg X 0.75 (% non-respirable) = 15.12 mg/day to GI tract; 15.12 mg X 0.75 (75% GI absorption) = 11.34 mg absorbed through GI tract. Total theoretical arsenic exposure would be:

5.04 (absorbed through lung)
11.34 (absorbed through GI tract)
16.38 mg/day total arsenic exposure per day.

If we assume the worker to be a 60-kg woman, exposure would be 16.38 mg / 60 kg = 0.27 mg/kg.

(b) Dermal Exposure

In order to determine the amount of arsenic which can enter the system, it is first necessary to determine how much arsenic is available on the surface of the treated wood. Several studies are available showing the amount of arsenic removed when dry, treated wood is wiped with a sponge, wet human hand, dry human hand, etc. Table 17 summarizes that data available on arsenic content of wood dust, splinters, etc. It is also desirable to determine the amount of arsenic which can be leached from wood when exposed to damp or flooded conditions. Table 18 summarizes the available data showing the amount of inorganic arsenic

which can be leached from treated wood. If the treated wood, e.g. as used in houses, is subjected to water from rain, flooding, etc., this additional arsenic would then be available and of importance when considering dermal exposure.

A representative exposure situation would be that resulting from the construction of homes using arsenic-treated wood. A typical activity would be the carrying, positioning, and nailing of treated plywood in the construction of floors of domestic dwellings. For purposes of this analysis the following assumptions are made:

1. The worker will work with four by eight foot sheets (32 sq ft) of arsenic-treated plywood;
2. The treated wood will be positioned and nailed at the rate of 7 sheets per hour;
3. The worker will work with the treated wood for 8 hours per day;
4. The worker will come in contact with 15% of the surface of the wood; and
5. The worker's hands will become damp with perspiration and could remove up to 0.529 mg/ft^2 arsenic (Dreher 1974).

Therefore, the worker will handle 224 sq ft of wood per hour and 1,792 sq ft during the course of an 8-hour day. If the worker's skin comes in contact with 15% of the wood, then the worker would contact 15% X 1,792 sq ft, or 268.8 sq ft of arsenic-treated wood per day. As shown by Dreher (1974) [see Table 17], a damp human hand can remove up to 0.529 mg arsenic per square foot of wood. If the worker contacts 268.8 sq ft, a total of 142.2 mg arsenic will come in contact with the skin per day. If the worker is wearing short pants and kneels on the wood during construction exposure would be higher. Assuming 10% absorption, 14.22 mg of arsenic will enter the body. If we assume the worker to be a 60-kg woman, exposure would then be calculated as 14.22 mg / 60 kg = 0.24 mg/kg resulting from contact with arsenic-treated wood. Fahlstrom et al. (1967) studied the leaching rate of CCA-treated wood samples. The authors found that treated wood leached with distilled water liberated 10.5% of its arsenic in two days. It is possible that dermal exposure to treated wood as discussed above would be significantly higher if treated wood was subjected to rain prior to or during construction.

Table 17. Arsenic Content of Dust, Splinters, Deposites, etc., from the Surface of Plywood Pressure-treated with Chromated Copper Arsenate (CCA) Wood Preservatives

Dwelling or Sample Number	Procedure Used to Assay for Arsenic- or Description of Sample	Amount of Arsenic Removed	Comment	Reference
A[a/]	Splinters	310 ppm >4,540 ppm 2,450 ppm	taken from treated wood in homes	NCMA (1976)
A	Wood Scraping	>190 ppm	"	"
A	Greenish salt-like Formation on Wood	3,050 ppm	"	"
B[a/]	Splinter	>460 ppm >420 ppm >340 ppm >440 ppm	"	"
B	White Deposit	>460 ppm	"	"
B	Dust Around knot Hole	>210	"	"
1	Splinter	12,000 ppm	"	Sleater and Berger (1977)
2	Splinter	15,000 ppm	"	"
4	Splinter	10,000 ppm	"	"
7[a/]	Splinter	10,000 ppm	"	"
8[a/]	Splinter	10,000 ppm	"	"

a/ Homes A and 7 are the same; Homes B and 8 are the same.
> Exceeded the limit of detection.

Table 17 (Continued)

Dwelling or Sample Number	Procedure Used to Assay for Arsenic or Description of Sample	Amount of Arsenic Removed (mg/sq ft)	Comment	Reference
-	Wood wiped with dry tissue	0.078-0.22	Dry wood	Dreher (1974)
-	Wood wiped with damp sponge	0.109-0.53		"
-	"	0.236-0.252	Wood soaked in quiescent water for 24 hours, air dried and wiped	"
-	Dry wood wiped with a dry human hand	0.0075-0.252	Wood - 2 wks old	"
-	"	0.005-0.0085	Wood - 2 yrs old	"
-	Dry wood wiped with a damp human hand	0.115-0.278	Wood - 2 wks old	"
-		0.255-0.529	Wood - 2 yrs old	"
-	Wood wiped with a dry human hand	0.0005-0.00095	Wood - 2 wks old Wood hosed down prior to wipe	"
-	"	0.005-0.015	Wood - 2 yrs old Wood hosed down prior to wipe	"

Table 17 (Continued)

Dwelling or Sample Number	Procedure Used to Assay for Arsenic or Description of Sample	Amount of Arsenic Removed (mg/sq ft)	Comment	Reference
-	Wood wiped with damp human hand	0.071-0.082	2-wk-old wood hosed down prior to wipe	Dreher (1974)
-		0.097-0.185	2-yr-old wood hosed down prior to wipe	"
7	Wood wiped with dry tissue	0.004	Taken from treated wood in home	Sleater & Berger (1977)
8	"	0.005	"	"
9	"	0.002	"	"
10	"	0.009	"	"
11	"	0.128	Taken from treated wood in home; whitish deposit where reinforcing rod meets wall	"

Table 18. The Amount of Arsenic Leached From Treated Wood

Type Preservative	Size of Wood Sample	Leaching Material	Duration of Test	Amount Leached	Comments	Reference
CCA (FDN)	3x5x5/8"	deionized water	1 day	5.3 ppm (0.12 mg/in)	0.6 pcf	Memo (1973)
			3 days	10 ppm (0.33 mg/in)		"
			7 days	13.3 ppm (0.30 mg/in)		
			28 days	45.5 ppm (1.03 mg/in)		
CCA (LP22)	3x5x5/8"	deionized water	1 day	3.0 ppm (1.03 mg/in)	0.4 pcf	Memo (1973)
			3 days	6.6 ppm (0.15 mg/in)		
			7 days	9.66 ppm (0.22 mg/in)		
			28 days	23.3 ppm (0.53 mg/in)		
Control	3x5x5/8"	deionized water	1 day	0.016 ppm (0.0004 mg/in)	control	Memo (1973)
			28 days	0.023 (0.0005 mg/in)		
CCA	1.5x5x0.125"	distilled water	2 days	0.4%	1.37 pcf	Fahlstrom et al. (1967)
			14 days	0.5%		
			2 days	3.1 %	1.22 pcf	
			14 days	8.2%		

Table 18 (Continued)

Type Preservative	Size of Wood Sample	Leaching Material	Duration of Test	Amount Leached	Comments	Reference
CCA	1.5x5x0.125"	distilled water	2 days	10.5%	0.796 pcf	Fahlstrom et al. (1967)
			14 days	20.75%		
			2 days	19.65%	1.29 pcf	
			14 days	35.%		
FCAP	2x1x0.5"	distilled water	3/8 hr	0.8%	0.124 pcf	McCarthy and Wilson (1957)
			22 7/8 hr	4.5%		
			3.95 days	10.0%		
			127.94 days	35.4%		
ACA	2x1x0.5"	distilled water	3/8 hr	6.5%	0.555 pcf	McCarthy and Wilson (1957)
			22 7/8 hr	18.8%		
			3.95 days	29.3		
			127.94 days	55.2%		
CCA	2x1x0.5"	distilled water	3/8 hr	0.0%	0.124 pcf	McCarthy and Wilson (1957)
			22 7/8 hr	0.1%		
			3.95 days	0.1%		
			127.94 days	2.8%		
CCA	60x75x100 cm	tap water	10 minutes scrubbed with brush - from round surface	0.07-0.39 mg/sq ft	0.94 pcf (approx)	Johanson and Dale (1973)
			10 minutes scrubbed with brush - from intact end area	1.15-2.44 mg/sq ft		

(4) Exposure to Individuals Living and/or Working in Dwellings Containing Arsenic-Treated Wood

Exposure to inorganic arsenic resulting from living or working in dwellings containing arsenic-treated wood occurs through the inhalation and dermal routes.

(a) Inhalation Exposure

Two studies [NCMA (1976) and Sleater and Berger (1977)] are available which discuss the amount of arsenic in air of dwellings containing arsenic-treated wood. Table 19 summarizes the results from these studies.

The NCMA (1976) study was conducted by an independent testing laboratory (Froehling & Robertson, Inc.) for the National Concrete Masonry Association. In this test the air of two homes was sampled using a high volume air sampler fitted with an 8 x 10 inch fiberglass filters. A volume of 4,860 cubic feet of air was passed through the filters at each location. The tests were conducted during normal living activities at each location. Residents were in and out, cleaning, cooking, etc. Chemical analysis of the samples was conducted using the silver diethyldithiocarbamate method.

The Sleater and Berger (1977) study was conducted by the National Bureau of Standards (NBS). In this study the

air of eight homes were sampled for arsenic content. The samples were collected using high volume air samplers fitted with fiberglass or paper filters of approximately 0.3 micron porosity. Between 200 and 2,000 cubic meter of air per site was sampled depending upon accessibility. Chemical analysis of the samples was conducted using the silver diethyldithiocarbamate method.

Both homes monitored by Froehling & Robertson (NCMA 1976) were also monitored by NBS (Sleater and Berger 1977), with markedly different results. Froehling & Robertson (NCMA 1976) monitored the houses on May 27, 1976, and recorded arsenic in air values of 0.6 to 3.6 mg/m^3. NBS monitored the houses in mid-December and mid-March of 1976 and recorded arsenic in air values of 0.002 to 0.031 ug/m^3.

In its report NBS commented concerning the markedly varying results of the NBS versus Froehling & Robertson. Of the differences in findings the authors note, "The disparity between the arsenic in air levels reported [refering to the NBS figures] cannot be explained at this time. It is clear the NBS has not confirmed the findings reported by NCMA, but neither has NBS refuted these findings." The authors note that a thorough cleaning of the residence prior to their visit could explain the lower levels of arsenic found by NBS. The results in Table 19 represent data from only seven

homes containing arsenic-treated wood. A survey of more treated homes could reveal much higher or lower levels of arsenic in air.

In order to calculate the theoretical arsenic exposure through inhalation, the following assumptions are made:

1. breathing rate to be 1.0 m^3/hr (resting mode) or 1.8 m^3/hr (active mode);

2. 100% of all arsenic in air will be respirable. A figure of 25% respirable was used when calculating inhalation exposure resulting from the cutting of arsenic-treated wood as discussed earlier. When cutting wood the fine particles of wood containing arsenic would become airborne but would probably not be completely respirable. In the case of a domestic dwelling, however, arsenic-containing dust would be of much smaller particle size and could therefore be respirable;

3. 100% of all respirable arsenic will be absorbed.

4. The resident of the dwelling will be present for 16-24 hours per day. It should be noted that, in the case of teratogenicity, the period of time during which the developing fetus is at risk may be

relatively short. Hood and Pike (1972), Hood et al. (1977; undated), etc., have demonstrated that arsenic exposure on one or two critical days of organogenesis will result in malformations. While it would be unreasonable to assume that a woman of child bearing age would remain in the arsenic treated home for 24 hours, day in and day out, it is not unreasonable to assume the woman to be present for any one continuous 24 hours period. If the developing fetus were to undergo a critical phase of organogenesis while the mother was present in a house containing arsenic-contaminated air it is not unreasonable to assume this exposure to continue for up to 24 hours.

Using the figure of 3.6 ug/m^3 (NCMA dwelling A) as a worst case situation and assuming the woman to be present in the house for 24 hours and that 16 of the 24 hours will be in the active mode (respiratory rate of 1.8 m^3/hr) and 8 hours in the resting mode (1.0 m^3/hr) exposure would be:

1.8 m^3/hr (active breathing rate) x 16 hours = 28.8, plus 1.0 m^3/hr (resting rate) x 8 hours = 36.8m^3/hr; 36.8 m^3/hr x 3.6 ug/m^3 (arsenic content in air) = 132.5 ug/24 hours or 0.132 mg/24 hours.

Assuming the resident to be a 60-kg woman, the resident would be exposed to 0.133 mg/60 kg = 0.002 mg/kg/day as a result of breathing arsenic-contaminated air.

Using the figure of 0.002 ug/m^3 (Sleater and Berger dwelling 8) as a best case situation and assuming the woman to be present in the house for 16 hours and that 8 hours of the 16 will be in the active mode (respiratory rate of 1.8 m^3/hr) and 8 hours in the resting mode (1.0 m^3/hr), exposure would be 7 x 10^{-7} mg/kg/day (trace).

Table 19. Arsenic in air of building constructed with wood pressure-treated with arsenical wood preservatives

Dwelling Number	Arsenic in Air (ug/m^3)	Comments	Reference
1	0.012	Treated wood exposed	Sleater and Berger (1977)
1	0.031	Treated wood covered	
2	0.016	Treated wood exposed	
2	--	Treated wood covered	
3	0.011-0.007	Masonry Constructed Foundation	
7	0.013	Treated Wood Exposed	
7	0.002	Treated Wood Covered	
8	0.019	Treated Wood Exposed	
8	0.002	Treated Wood Covered	
9	0.002	Treated Wood Exposed	
9	0.003	Treated Wood Covered	
10	0.005	Treated Wood Covered	
10	--	Treated Wood Covered	
11	0.003	Treated Wood Exposed	
11	0.003	Treated Wood Covered	
1-11	0.002	Average arsenic content of outside air	
A*	3.6	No Comment	NCMA (1976)
B*	0.6	No Comment	

a/ In some cases both NBS and NCMA examined the same dwellings; home A and 7 are the same dwelling, as are B and 8.

Table 20. Arsenic in dust of homes containing arsenic-treated wood

Dwelling Number	Arsenic Content of dust (ppm)	Comment	Reference
1	211	Exposed Plywood	Sleater and Berger (1977)
1	2	Covered Plywood	
3	28-27	Masonry Construction	
4	53	Covered Plywood	
6	1267	Exposed Plywood	
6	7	Covered Plywood	
7	123-354	Exposed Plywood	
8	61-69	Exposed Plywood	
9	138	Exposed Plywood	
0	18	Covered Plywood	
11	10	Exposed Plywood	
A	18 32 >440	No Comment	NCMA (1976)
B	>210 1640	No Comment	
-	11 21 13 17 23	From vacuum cleaner sweepings	Klemmer et al. (1975)

(b) Dermal Exposure

Dermal exposure could arise from direct contact with treated wood, or contact with arsenic-containing dust produced from or by treated wood.

(i) Direct Contact with Treated Wood

Tables 17 and 18 summarize relevant data concerning the amount of arsenic available in splinters, on the surface of treated wood, and the amount of arsenic which can be leached by water. The highest rates are listed in Table 21.

Table 21.

Source	Amount of arsenic	Comment	Reference
Greenish salt-like formation on wood	3,050 ppm	taken from treated wood in a home	NCMA (1976)
Splinter	15,000	"	Sleater and Berger (1977)
Wood wiped by damp human hand	0.5.9 mg/ft^2	wood 2 yrs old	Dreher (1974)
Treated CCA wood soaked in deionized water	148.3? mg/ft^2	wood soaked for 28 days	Memo (1973)
"	43.2 mg/ft^2	wood soaked for 7 days	"

The Agency assumes that, based on the above data, a homeowner coming into contact with treated wood after flooding would experience the greatest potential exposure. It is conceivable that a home basement could be subjected to flooding for long periods of time. When pressure-treated wood, such as CCA-treated wood, is used in the construction of foundations, a polyethylene film is installed between the soil and the treated wood foundation. The American Wood Preservers Institute (AWPI 1976) says of this polyethylene film, "It should be understood that the polyethylene film serves as a water deflector and is not intended as a water proof membrane. The film will direct ground water downward and away from the wall to the drainage area below. Design of the AWWF (all weather wood foundation) anticipates that the plywood wall may become wet in some cases...."

For purposes of analyzing potential arsenic exposure resulting from the flooding of home basements constructed of arsenic-treated wood, the following assumptions are made:

1. The basement will be continuously flooded for 7 days;
2. The basement will be 30 feet long, 30 feet wide, and 8 feet high and will be flooded to a depth of 4 feet. This will result in a total of 480 sq. ft. exposed to flood waters;
3. When the flood water recedes 75% of the leached arsenic will be deposited onto the walls or floor;
4. The homeowner will clean the basement after the flood and will require not more than one day to complete the task;
5. 10% of the residual arsenic will come into contact with the skin; and
6. 10% of all arsenic coming into contact with the skin will be absorbed.

Therefore, 480 sq ft (amount of wood subjected to leaching) X 43.2 mg/ft^2 (amount of arsenic leached per sq ft [Memo 1973]) = 20,736 mg leached from wood; 20,736 mg X 0.75 (% of leached arsenic deposited on to walls or floor = 15,552 mg arsenic; 15,552 mg X 0.1 (% coming in contact with the skin) = 1,555 mg arsenic; 1,555 mg X 0.1 (% absorption) = 155 mg arsenic absorbed. Assuming the person at risk to be a 60-kg woman, exposure would be 155 mg / 60 kg = 2.59

mg/kg resulting from exposure to arsenic-treated wood after flooding.

If flooding were to continue for 28 days exposure would be 8.8 mg/kg.

(ii) Contact with Arsenic-Containing Dust Produced from or by Treated Wood

Table 20 summarizes the relevant data concerning the amount of arsenic available in the dust of homes containing arsenic-treated wood. The Working Group assumes that the homeowner can be dermally exposed with up to 10 grams of dust during the sweeping of arsenic-laden dust, that this dust will contain 1,640 ppm arsenic (Table 20), and that 10% of all arsenic will be absorbed. Therefore, 10 gm of dust X 1.64 mg arsenic/gm of dust (equivalent to 1,640 ppm) = 16.4 mg arsenic coming in contact with the skin. Assuming 10% is absorbed, the homeowner will assimilate 1.64 mg of arsenic. If the resident is a 60-kg woman, the dosage of arsenic would 1.64 mg / 60 kg = 0.03 mg/kg due to dust from arsenic-treated wood.

Total theoretical dermal exposure will be the sum of direct contact with treated wood and contact with dust containing arsenic produced from or by treated wood:

3.45 mg/kg (from flooded basements)
0.03 (contact with dust)

3.48 mg/kg total theoretical dermal exposure.

C. Summary

Table 22 summarizes the available information concerning exposure of a 60-kg woman to arsenic resulting from the use of inorganic arsenical pesticides. The only registered use for sodium pyroarsenate, ammonium arsenite, and arsenic pentoxide is for wood preservation situations; the levels of exposure to these forms of inorganic arsenic are listed under this category. Inorganic arsenical wood preservatives are commonly sold in three formulations (ACA, CCA, and FCAP). Exposure estimates for inorganic arsenical wood preservatives, as detailed in Table 22, were not calculated for each formulation/use situation; however, exposure estimates would be comparable for each use situation.

Human exposure as discussed in this document refers only to exposure resulting from the use of inorganic arsenical pesticides. Other sources of exposure to inorganic arsenic (i.e. smelters, coal-burning plants, volcanic debris) which contribute to the total exposure of the human population have not been discussed. This total exposure to inorganic arsenic would be higher than the exposure from pesticide use alone.

Table 22. Summary of the Total Theoretical Exposure To Inorganic Arsenic Resulting From the Use of Inorganic Arsenical Pesticides

Chemical or Use Situation	Total Theoretical Exposure (for a 60-kg Woman in mg/kg)
Agricultural Situations	
Sodium Arsenite	5.5
Arsenic Acid	2.72
Sodium Arsenate	0.65
Copper Acetoarsenite	0.32
Calcium Arsenate	0.17
Arsenic Trioxide	0.68
Lead Arsenate	0.66
Wood Preservative Situations (Sodium Pyroarsenate (Arsenic Trioxide, Arsenic Pentoxide, Sodium Arsenate, Arsenic Acid, and Ammonium Arsenite)	
Large Industrial Situations	
Inhalation Exposure	0.05
Dermal Exposure	0.57 - 9.0
Intermittent Application to Wood During On-Site Construction	
Dermal Exposure to Ammonium Arsenite	1.1
Exposure From Handling, Sawing, Fabricating, etc, of Treated Wood Products	
Inhalation Exposure	0.27
Dermal Exposure	0.24
Exposure To Individuals Living or Working In Dwellings Containing Arsenic-Treated Wood	
Inhalation Exposure	$7X10^{-7}$ - 0.002
Dermal Exposure	0.03 - 2.59

IV. SUMMARY OF SCIENTIFIC EVIDENCE TO SUPPORT REBUTTABLE PRESUMPTION

A. Relation Between Arsenate and Arsenite

The inorganic arsenical pesticides discussed previously all have the element arsenic in common. Studies in the following sections describe oncogenic, mutagenic, teratogenic, and other adverse effects of inorganic arsenic salts and/or their metabolites. The Working Group contends that the effect(s) observed are typically due to arsenic from the arsenate or arsenite ions released upon dissociation of the salt in various aqueous systems. The Agency notes that inorganic arsenic salts (i.e. sodium arsenate, sodium arsenite) will, to a greater or lesser extent, dissociate.

40 CFR 162.11(a)(3) provides that "A rebuttable presumption shall arise if a pesticide's ingredient(s), metabolite(s), or degradation product(s) meet or exceed any of the...criteria for risk." The term "degradation product" is defined by 40 CFR 162.3(l) to mean "a substance resulting from the transformation of a pesticide by physico-chemical, or biochemical means," and the term "metabolite" is defined by 40 CFR 162.3(w) to mean "any substance produced in or by living organisms by biological processes and derived from a pesticide." The liberated arsenate and

arsenite ions of the inorganic arsenicals discussed in this document can be considered ingredients of the arsenical pesticides or are encompassed by the 40 CFR definitions of "degradation product" and/or "metabolite."

For purposes of issuing an RPAR, the Agency has made the following determinations:

1. If any form of trivalent inorganic arsenic (arsenite) produces any given chronic effect, it must be assumed that all forms of trivalent inorganic arsenic are capable of producing similar effects, because they all dissociate to the common arsenite ion. This rationale will also prevail for pentavalent inorganic arsenicals (arsenates). The Agency has determined that when a rebuttable presumption is issued for any one pesticide containing arsenate or arsenite, an RPAR will be issued for all pesticides containing arsenate or arsenite, respectively;

2. In addition, as discussed in Section I.J, there is strong evidence that trivalent forms of inorganic arsenic will be oxidized to a pentavalent state in humans, other animals, and the environment. The pentavalent forms of arsenic would be considered degradation products or metabolites (biologically mediated) of trivalent arsenic (arsenites) and, therefore, the trivalent form of arsenic will be subject to regulation based on information from studies on its pentavalent metabolites. The Agency assumes, therefore, that the effects observed in tests using trivalent

arsenites may be due to arsenite, arsenate, or both. The EPA has determined that when an RPAR is issued for any one pesticide containing arsenic in the pentavalent form (arsenate), an RPAR will be issued for all arsenical pesticides containing arsenite, as well as for those containing arsenate; and

3. As discussed in Section I.J, there is also evidence that pentavalent arsenic (arsenate) can be converted to trivalent arsenic *in vivo*. Under these conditions, arsenite would be a metabolite of arsenate and therefore arsenate will be subject to regulation based on information from studies on its trivalent metabolites. The Agency has determined that when an RPAR is issued for any one pesticide containing arsenic in the trivalent form (arsenite), an RPAR will be issued for all arsenical pesticides containing arsenate, as well as for those containing arsenite.

B. Chronic Toxicity

The following adverse effects of inorganic arsenic have been found to exceed the criteria for issuance of a rebuttable presumption.

(1) Oncogenic Effects

40 CFR Section 162.11(a)(3)(ii)(A) provides that a rebuttable presumption shall arise if a pesticide's

ingredient(s) "(i)nduce(s) oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation, or dermal exposure...." Section 162.3(bb) defines the term oncogenic as "the property of a substance or a mixture of substances to produce benign or malignant tumor formation in living animals." Epidemiological studies, in conjunction with confirmatory animal tests, provide the best evidence that an agent is a human carcinogen. Albert et al. (1977) noted that "of the chemical agents that are generally accepted to have produced human cancer, all but one [arsenic], produced a carcinogenic response in rats and/or mice...in the same organ as in humans when tested by the appropriate route of exposure." Since arsenic is unique in this respect (Frost 1967; NAS 1977b), the Agency has relied primarily on human epidemiology studies in its assessment of the oncogenicity of inorganic arsenicals.

The information and background data included in the following portion of the position document have been taken from the EPA Carcinogen Assessment Group's Preliminary Report on Inorganic Arsenic (EPA 1978). Studies discussed in this report show that both the trivalent (arsenite) and pentavalent (arsenate) forms of inorganic arsenic act as oncogenic agents. For this reason the

Agency concludes that, <u>without exception</u>, a rebuttable presumption against registration exists for <u>all</u> inorganic arsenical pesticides based on the oncogenic effects criteria. The various forms of inorganic arsenic are enumerated in Section I.A.

Both the National Academy of Science (NAS 1977b) and the Occupational Safety and Health Administration (OSHA 1978) have conducted extensive reviews of the literature concerning the oncogenicity of arsenic. The reader is referred to the NAS and OSHA publications for additional epidemiological studies concerning the oncogenicity of arsenic.

Chronic exposure of humans to inorganic trivalent arsenic from drinking water, medicinal preparations, pesticides, non-ferrous metal smelters, and a gold mine operation has resulted in abnormal pigmentation, keratosis, and skin cancer as well as cancer of the lung and liver in some cases. Chronic exposure of pesticide-manufacturing workers and orchard workers using lead arsenate has resulted in excess incidences of lung cancer. Although concurrent exposure to other chemicals may have contributed to the observed incidence of cancer, the weight of evidence suggests that chronic exposure to either trivalent or pentavalent arsenic can induce cancer. This conclusion is supported by toxicological and mutagenesis studies [see Sections I.J and IV.B.(2)] which show that the two forms of arsenic have similar effects, although trivalent arsenic is more potent than the pentavalent form.

A large body of literature shows that chronic arsenic exposure results in symptoms of abnormal skin pigmentation, keratosis, circulatory disorders leading to gangrene, squamous cell carcinomas, and basal cell epitheliomas of the skin as well as bronchial carcinoma and liver vascular sarcomas (angiosarcomas). These various symptoms result from several exposure situations, summarized below; the valence state of the arsenic compound involved and concomitant exposure to other agents are also considered.

(a) Medical Exposure

Comprehensive reviews by Neubauer (1947), the National Academy of Sciences (NAS 1977b), and Stellman and Kabat (1978) document the occurrence of skin cancer and its early symptoms in patients treated for psoriasis and other conditions with potassium arsenite (Fowler's solution) in the United States before 1950. Since pentavalent arsenic salts are generally not effective therapeutic agents, little information is available concerning their chronic effects from this usage pattern. The evidence indicates that continued administration of potassium arsenite causes circulatory disturbances of the skin, hyperpigmentation, keratosis, skin cancer and, rarely, liver (hepatic) angiosarcoma. Neubauer (1947) found that, in 143 patients with medical skin cancer who had been treated with trivalent arsenic, the latent time between first administration of therapy and the onset of superficial epithelioma ranged between 3 and 40 years, with an average of 18 years. The epitheliomas typically developed on patches of keratotic areas of skin, sometimes after the

keratosis had been present for several years; but they also developed on healthy areas of the skin and on patches of psoriasis.

(b) Drinking Water Exposure

Chronic arsenical poisoning, including skin cancer and a gangrenous condition of the hands and feet called Blackfoot's disease, has occurred in several communities exposed to arsenic in drinking water. The best-documented instance of such arsenical poisoning is in Taiwan (Yeh et al. 1968; Yeh 1963; Yeh 1973; Tseng et al. 1968; Tseng 1977). In a house-to-house survey of 40,421 people in 37 villages along the southwest coast of Taiwan, Tseng et al. (1968) found that the prevalence of skin cancer, hyperpigmentation, and keratosis each correlated with the arsenic content of the water. The highest range of concentrations was greater than 0.6 ppm, and the lowest range was less than 0.29 ppm. From a general survey of these inhabitants, the prevalence rate for arsenical skin cancer was 10.6 per 1,000 residents in 1965. The skin cancer rate for well water containing >0.60 ppm was 21.4 per 1,000 people, while at <0.29 ppm it was 2.6.

The sources of drinking water for these areas were deep artesian wells, used from 1910/20 until 1966, when a tap water supply was installed. The concentrations of arsenic in the water ranged from 0.01 to 1.82 ppm (median range was 0.4 ± 0.6 ppm). The chemical form of arsenic in the water was not determined, but it may have been either trivalent (because the well-water is probably anaerobic) or

a methylated arsine (because the authors observed a combustible gas [perhaps methane] bubbling from the water storage tanks). Attempts to measure the arsenate to arsenite ratio of the water have been confined to measurements in a United States laboratory of a sample shipped from Taiwan with no special precautions to preserve the speciation occurring at the collection point (Irgolic 1978). The arsenate to arsenite ratio in two samples was 10 to 1 and 7 to 1, with a total arsenic concentration of approximately 0.6 ppm. Since the native water is likely to be anaerobic and since oxidation probably took place to some degree in transit to the analysis laboratory, the ratio in the natural water is probably less than 10 but cannot be assessed at this time. Therefore, from the best available information, people in that region of Taiwan could have been exposed to both trivalent and pentavalent arsenic compounds.

Assessing the Taiwan situation is more complex than simply identifying the two oxidation states of arsenic, as reported recently by Lu et al. (1975, 1977a, 1977b). These workers have observed non-arsenical fluorescent compounds in water samples from the areas where Blackfoot disease is endemic and have identified one of the fluorescent components as an alkaline hydrolysate of ergotamine, lysergic acid, or a related compound (Lu et al. 1977b). They have also shown that one of the fluorescent components produced abnormalities in developing chick embryos (Lu et al. 1977a). It is not known whether ergotamine was the compound that produced these abnormalities.

Two other areas of high levels of arsenic in drinking water are the Cordoba province of Argentina (Bergoglio 1964) and the town of Reichenstein in Selesia (Neubauer 1947). The former is a large farming area with natural geologic deposits of arsenic-containing rock, and the latter is adjacent to a former gold mine with arsenic-containing ores. Skin cancer and other disorders were observed in a large number of people in the areas of Cordoba with high levels of arsenic in the water. The arsenic contamination of ground water in Reichenstein is believed to be responsible for the widespread symptoms in the local population of melanosis and keratotic warts on hands and fingers, perforation of the nasal septum, mouth ulcers, gastrointestinal tract disorders, and skin cancer. The chemical form of arsenic in the drinking water was not stated in these reports.

(c) Exposure in Smelters

Ore containing non-ferrous metals (copper, zinc, and lead) also has high concentrations of arsenic that must be removed in order to produce quality metals. At the smelters producing these metals, arsenic dust is found in the air largely in the form of arsenic trioxide [As_2O_3] (NAS 1977b) or perhaps arsenic sulfides [As_2S_2 or As_2S_3] (Smith et al. 1976). But, as emphasized by Arsenault (1977), the workers are concurrently exposed to sulfur dioxide (Lee and Fraumeni 1969), as well as to sulfates and sulfites (Smith et al. 1976). Occupational

studies of smelter workers have been reported in the United States by Pinto and Bennett (1963), Lee and Fraumeni (1969), Milham and Strong (1974), and Pinto et al. (1977), and in Japan by Kuratsune et al. (1974) and Tokudome and Kuratsune (1976). Excess rates of lung cancer among the employees with the highest exposure to arsenic have been found consistently in these studies, although the early Pinto and Bennett study (1963) did not show a significant difference between exposed and control groups. The consistency of these findings in different smelter environments leaves little doubt that arsenic trioxide is at least a major contributor to the incidence of lung cancer in heavily exposed workers. Lee and Fraumeni (1969) have shown that lung cancer rates are also correlated with sulfur dioxide exposure, but there were no populations which had exclusive exposure to either of the agents. Lung cancer mortality among patients in a southern Rhodesian gold mining district has been studied by Osburn (1969). He found that of 37 people who died over 15 years, 13 also had hyperkeratosis characteristic of chronic arsenical poisoning. All but one patient had known exposure to dust containing arsenic from the mines.

(d) Exposure to Inorganic Arsenical Pesticides

Both trivalent and pentavalent arsenic are used in pesticide products. Reports of skin, lung, and liver

cancer in people exposed during the manufacture and use of these compounds have been published. In manufacturing plants an excess of respiratory cancer was reported by Ott et al. (1974), Ott (1976), and Hill and Faning (1948). The use of inorganic arsenical pesticides has been associated with cancer of several organs in German and French vintner workers by Roth (1956, 1957a, 1957b, 1957c, 1958), Denk et al. (1969), Thiers et al. (1967), Braun (1958), von Pein (1943), Galey et al. (1963), and Petres and Hagedorn (1975).

(i) Exposure in Pesticide Manufacturing Plants

A study of factory workers in the Dow Chemical Company's pesticide manufacturing plant (Ott et al. 1974) showed excess rates of respiratory and lymphatic cancer. The authors found that the rate of respiratory cancer correlated with the inorganic arsenic exposure level. Although there was minor exposure in a building where the arsenic trioxide starting material was loaded into the equipment, the workers were predominantly exposed through bagging to finished calcium and lead arsenate products. Production of arsenic compounds over the entire 37 years of operation was estimated to be 59% lead arsenate, 34% calcium arsenate, 5% copper acetoarsenite, and 2% magnesium arsenate.

These effects can therefore be ascribed to pentavalent, rather than to trivalent arsenic. Exposure to powdered sulfur and dry lime sulfur also occurred (Ott et al. 1974).

In a British factory manufacturing a sodium arsenite sheep dip compound, Hill and Faning (1948) observed a high rate of lung and skin cancer among workers directly handling the compound.

(ii) Exposure via Use of Pesticides

Inorganic arsenic pesticides were used on grapes in France and Germany between the early 1920s and 1942. Roth (1957b) reported the occurrence of chronic arsenical poisoning, including sequelae of skin and lung cancer, in French and German vintners. (These same sequelae of skin and lung cancer have also been observed in patients medicinally treated with inorganic arsenic.) In addition, a rare form of liver cancer, hemangio-endothelioma, was observed in the vintners. The most complete description of the cases is in a series of papers by Roth concerning the Mosel River Valley wine-growing area, but chronic arsenical poisoning also occurred in the upper Baden Valley, where it was called the "Kaiserstuhl Sickness" (Petres and Hagedorn 1975), and in the Rhine, Pfalz, and Wachau regions in Germany and the Beaujolais region in France (Galey et al. 1963; Thiers et al. 1967).

As Roth (1958) points out, not all wine-growing regions in Germany used inorganic arsenical insecticides because the particularly voracious insect that was susceptible to arsenic poisoning was not present everywhere. This insect is Cochylis (or Clysia) ambiguella, also known as the hay worm and sour worm (Thiers et al. 1967; Stellwaag 1928). It has two generations per growing season. The first caterpillars appear in June when the hay is harvested, and the second generation appears in late summer. A detailed description of these insects is given by van Emden (1936).

A dust of calcium arsenate, applied every two weeks in early summer, was particularly effective against hay and sour worms (Stellwaag 1928). This mixture contained calcium and As_2O_5 (pentavalent arsenic) with an arsenic concentration of 10 to 15%, and was called Sturm's Dusting Material by some (Frohn 1938). This technique was not popular for long because of the extremely acute reaction of the vintners to the dust, but it was used in cases of severe infestation. Another arsenic preparation, applied as a spray, was a mixture of copper acetoarsenite (or paris green), also known by the trade names of "Selesia Green" and "Urania Green," which is a copper-lime solution (Stellwaag 1936). The copper-lime solution alone controls the common fungus

Peronspora, and the combination spray (including paris green) is effective against both the fungus and the hay and sour worms. The vintners were therefore exposed to both trivalent arsenic, as copper acetoarsenite, and pentavalent arsenic, as calcium arsenate, during the growing season.

A second, and perhaps major source of vintner exposure to inorganic arsenic was by drinking the "house wine," prepared from the second pressing of grape skins. Stellwaag (1928) states that, during the fermentation process, a large fraction of the arsenic was removed from the liquid by transformation into arsenic sulfide, which was subsequently absorbed by the yeast. This form of arsenic should, therefore, appear in the "house drink," which was consumed in quantities of 1 to 3 liters per day during the warm season. Crecelius (1977a) has shown that, during the fermenting of grape juice, the final ratio of trivalent to pentavalent arsenic was about 4 to 1, regardless of whether the starting material was all trivalent or all pentavalent, and that the fermenting process tends to convert arsenic of both valences predominantly to the trivalent form.

In summary, the inorganic arsenic responsible for the chronic poisoning and cancer in the vintners was a complicated mixture of pentavalent and trivalent compounds, perhaps combined with sulfur.

The Wenatchee Valley in central Washington is another site where arsenical pesticides have been used for many years in the form of lead arsenate in apple orchards. Nelson et al. (1973) found no excess rate of lung cancer in orchardists, but the National Institutes of Occupational Safety and Health (NIOSH 1975) recently re-examined the mortality statistics in the state of Washington and found a statistically significant increase of respiratory cancer.

An epidemiological study of carpenters in Hawaii, who were exposed to pentavalent arsenic wood preservatives for an unknown period of time, was published by Budy and Rashad (1976). The rate of respiratory cancer among carpenters who died between 1970 and 1973, as compared to matched non-carpenter control groups, was not significantly different than the corresponding rate for those who died between 1948 and 1952. In the earlier years, about 5% of the wood was treated with arsenic, whereas in the early 1970s about 50% of the wood was so treated. Uncertainty about the degree and duration of exposure makes this study difficult to interpret.

(e) Summary of Human Exposure Studies

Chronic exposure of humans to sufficiently high doses of arsenic in several situations has produced abnormal

pigmentation, keratosis, and sk n cancer as wel as cancer of lungs, liver, and perhaps her sites The adverse effects resulting from this exposure have been consistently the ·ame as those produced by medicinal use of potassium arsenite; and, with two exceptions, the exposures have all involved trivalent arsenic. Therefore, it is extremely likely that trivalent arsenic is a human oncogen. Two situations of arsenic exposure, in which the predominant form of arsenic is pentavalent, have produced lung cancer; these are the Dow pesticides manufacturing plant and the lead arsenate exposures in Wenatchee, Washington. Although it is difficult to rule out other chemicals as the causative agents, these two situations furnish ample cause for concern that pentavalent arsenic may also be oncogenic.

(f) In Vitro Studies Suggestive of Oncogenic Potential

Although *in vivo* animal studies have not yet confirmed inorganic arsenic to be oncogenic, several positive *in vitro* cell transformation studies have been published. For certain cell lines, *in vitro* transformation from primary (normal, diploid) cells to cancerous ones is a good indicator of the carcinogenic potential of a compound. The best validated test systems are the Syrian hamster embryo cell system of Dr. R. Pienta (NCI) and the Chinese hamster ovary cell system used by Dr. DiPaolo (NCI).

These are short-term testing systems for oncogenicity and produce few, if any, false positives.

Casto et al. (1976) showed that sodium arsenite induced the transformation of Syrian hamster fetal cells that were seeded to form colonies. Arsenite also enhanced the frequency of simian adenovirus (SA7) transformed foci. Spontaneous transformations were not observed in the controls. The increase in SA7 transformation frequency was not due to selective toxicity, since there was a 2- to 3-fold increase in treated cultures in the number of foci per plate.

Casto (Letter 1977a) elaborated on this work (described above) with data that supported the conclusion of *in vitro* transformation of these hamster embryo cells by sodium arsenite. At a concentration of 1.2 ug/ml, transformation was enhanced 2.4 times over the background level (significant at 5% level). The responses were dose-related.

Casto et al. (1977) assayed Syrian hamster embryo cells for enhancement of viral transformation by an inorganic salt of arsenic (sodium arsenite). Hamster cells were exposed to arsenic either 18 hours before or 5 hours after the addition of simian adenovirus (SA7). Survival was determined by cloning methods, and viral transformation

was detected by focus assay. There was a significant increase in the relative transformation frequency and in the absolute number of SA7 foci per plate. This indicates the increase was not due to selective toxicity. Enhancement occurred at concentrations that did not affect cloning efficiency.

Moore (1976) used Fischer rat embryo cells infected with Rauscher leukemia virus and a cloned cell line of mouse BALB/3T# to assay for in vitro cell transformation by arsenic trioxide. A positive control (3-methylcholanthrene) was used. The results for the rat and mouse cell systems were negative. The Fischer rat embyro system is not validated.

(g) Conclusion

EPA concludes that sufficient epidemiological data exists to show that both trivalent inorganic arsenic (arsenite) and pentavalent inorganic arsenic (arsenate) act as oncogenic agents in humans. This position is in agreement with that of the Occupational Safety and Health Administration (OSHA 1978) who conclude "(t)hat there is virtually no dispute as to the carcinogenicity of trivalent arsenic," and that, on the basis of the available evidence, "pentavalent arsenic should be regulated as a human carcinogen." Section I.J presents strong evidence of the ability of inorganic arsenic to interconvert in vivo (arsenite to arsenate and arsenate to arsenite). As discussed in Section I.J, this interconversion makes

trivalent arsenic a metabolite of pentavalent arsenic and pentavalent arsenic a metabolite of trivalent arsenic.

Therefore, in the case of pentavalent inorganic arsenic, the Agency stresses that the RPAR is being issued for two reasons: 1) that pentavalent arsenic is an oncogen and 2) that pentavalent arsenic is reduced in vivo to trivalent arsenic, which also acts as an oncogen. The Agency concludes that either characteristic of pentavalent arsenic is sufficient criterion for the issuance of an RPAR.

The basis for issuance of an RPAR for trivalent inorganic arsenic is similar: 1) that trivalent arsenic is an oncogen and 2) that trivalent arsenic is oxidized in vivo to pentavalent arsenic, which also acts as an oncogen.

(2) Mutagenic Effects

40 CFR Section 162.11(a)(3)(ii)(A) provides that "A rebuttable presumption shall arise if a pesticide's ingredient(s), metabolite(s), or degradation product(s) ...induces mutagenic effects, as determined by multi-test evidence."

Studies discussed in this report (and summarized in Table 23) show that both trivalent (arsenite) and pentavalent (arsenate) forms of inorganic arsenic can act as mutagenic agents. The EPA concludes that these studies represent multi-test evidence and that, without exception,

Table 23. Summary of Studies of Inorganic Arsenic Mutagenicity.

Study	Author(s)	Chemical(s)	Cell Type	Effects
1. Point Mutation	Ficsor and Lo Piccolo 1972	sodium arsenate	E. coli	No reversions induced
	Moore 1976	arsenic trioxide	S. typhimurium	Negative Ames' test (without metabolic activation)
	Nishioka 1975	sodium arsenite	E. coli	Positive mutagen (induced tryptophan reversions).
	Casto (Letter 1977a)	sodium arsenite	hamster lung cells	Positive mutagen - increased the frequency of mutation in 8-azaguanine treated cells.
2. Chromosomal Effects In vitro Cytogenetics	Oppenheim and Fishbein 1965	potassium arsenite	human peripheral leukocytes	Chromosome gaps, breaks, translocations, dicentrics, ring forms were found. Cell division was supressed and there was an increased number of broken metaphase plates.
	Paton and Allison 1972	sodium arsenite	human diploid fibroblasts	Chromatid breakage
		sodium arsenite	human diploid fibroblasts and leukocytes	Chromatid breakage > Effect higher with sodium arsenite
		sodium arsenate		Chromatid breakage > Effect higher with sodium arsenite
In vivo Cytogenetics	Petres et al. 1970, 1977	unspecified	human lymphocytes (from psoriasis patients and wine-growers)	Aneuploidy, chromatid breaks, acentric fragments, and dicentric chromosomes occurred more frequently in cells from persons exposed to arsenic than in the controls.
Dominant Lethal Effects	Sram and Bencko 1974	sodium arsenite	ICR-SP mice	An acute oral dose (250 mg/kg) given to the male produced negative results. Chronic application over 4 generations of males at 10 mg/liter of drinking water gave positive results (increase in overall dominant and preimplantation lethality). A subtoxic dose (100 mg/liter) gave no change in frequency of dominant lethality or male fertility.
3. DNA Damage	Burgdorf et al. 1977	potassium arsenite	human lymphocytes	There was a higher rate of sister chromatid exchange in lymphocytes from patients who had used Fowler's solution compared to lymphocytes from unexposed controls.
4. DNA Repair (microbial)	Rossman et al. 1975, 1977	sodium arsenite	E. coli (uv-exposed)	There was decreased mutation frequency of irradiated cells that were excision-repair deficient. There was decreased survival rate of uv-exposed cells (wild type, excision-repair deficient). No effect was found on strain deficient in post - replication repair.
	Nishioka 1975	sodium arsenate sodium arsenite	Bacilus subtilis	Both chemicals were positive mutagens. The effect was stronger with sodium arsenite.
(mammalian)	Jung et al. 1969	sodium arsenate	human epidermal cells (exposed to xenon lamp)	DNA dark repair activity decreased in presence of sodium arsenate.
	Jung and Trachsel 1970	sodium arsenate	human epidermal cells (exposed to xenon lamp)	DNA synthesis (reduced mitotic index) and DNA dark repair were both inhibited in presence of sodium arsenate.
5. Metabolic and Cellular Toxicity Effects (in vivo)	Skipper et al. 1951	potassium arsenite	mice	There was reduced incorporation of ^{14}C-formate (precursor of mouse DNA-purines) into nucleic acid purines.
	Sawada and Rebhun 1969	sodium arsenite	annelid worm eggs	There was inhibition of the formation of the mitotic apparatus (before fertilization).
(in vitro)	Tsuda 1974	sodium arsenate	mouse fibroblasts	The indices for prophase, metaphase, and mitosis increased, but decreased for telophase.
	Petres and Hundeiker 1968	sodium arsenate	human peripheral lymphocytes	The number of mitoses was reduced to 1/5 the expected number. Chromosome pulverization occurred consistently.
	Petres and Berger 1972	sodium arsenate	same	At high concentrations, the mitotic index decreased. There were metaphase plate pulverizations at low doses. There was inhibition of ^{3}H-thymidine incorporation into DNA.
	Petres et al. 1974	sodium arsenate	same	There was inhibition of ^{3}H-thymidine and ^{14}C-thymidine incorporation into DNA at lower doses than inhibition of ^{3}H-uridine and ^{14}C-uridine into RNA.
	Petres et al. 1975	sodium arsenate	same	After one hour of exposure, there was increasing inhibition of ^{14}C-thymidine incorporation into DNA.
	Baron et al. 1975	sodium arsenate	same	There was inhibition of ^{14}C-TTP into DNA and ^{14}C-UTP RNA. There was also inhibition of incorporation of labeled alanine and leucine into cellular proteins.

a rebuttable presumption against registration exists for all inorganic arsenical pesticides based on the mutagenic effects criterion. The various forms of inorganic arsenic are enumerated in Section I.A.

(a) Studies Relevant to Rebuttable Presumption

(i) Point Mutation Studies

Nishioka (1975) examined the mutagenic effect of sodium arsenite on strains of *E. coli* by reversion studies. The *E. coli* strains used were WP_2 (trp^-, $uvrA^+$, $recA^-$); WP_2 uvrA (trp^-, $uvrA^-$, $recA^+$); and CM_{571} (trp^-, $uvrA^+$, $recA^-$). These strains were exposed to sodium arsenite concentrations ranging from 1.6×10^{-4} to 8.0×10^{-4} M. Reversions were induced in all but the CM_{571} strain of *E. coli*.

Casto (Letter 1977a) demonstrated that sodium arsenite was a point mutagen. Chinese hamster lung diploid cells (*Cricetulus griseus*, DON line) were grown in THAG medium. Sodium arsenite was added to the culture for 18 hours and then removed by adding fresh plain medium. After a 48-hour expression time, 10 ug/ml of 8-azaguanine was added. The culture medium was replaced with fresh medium containing 8-azaguanine every 48 hours for 20 to 25 days of incubation. For mutation assays the cells were fixed and stained. The cells for survival assays were grown in medium with the

arsenic and then placed in plain medium. The cells were later fixed and stained and surviving colonies were counted.

The frequency of mutation was expressed as the number of 8-azaguanine resistant colonies per 10^6 surviving cells. The spontaneous mutation frequency of this cell line was less than 1 mutant per 10^6 surviving cells. Without sodium arsenite, there were 0.45 8-azaguanine resistant colonies per 5×10^5 cells (mutation frequency of 0.9 colonies per 10^6 surviving cells). At 5 ug/ml of sodium arsenite, there were 9.6 resistant colonies per 5×10^5 cells (mutation frequency of 65.0 mutant colonies per 10^6 surviving cells).

Fiscor and Lo Piccolo (1972) tested an ant poison, containing 3% sodium arsenate, for mutagenicity by bacterial-plate assay. The compound (dosage not given) was tested for its ability to revert two *E. coli* lac^- amber mutants (NG 422, YA 482) to the lactose-fermenting phenotype, and to revert a cys^- (cysB12) and two leu^- (AP517, 5BU504) auxotrophs to prototrophy. All five strains had a low frequency of spontaneous reversions, and each was reversible by the positive control (known mutagen) N-methyl-N'-nitro-N-nitrosoguanidine. The results were negative.

Moore (1976) used the Ames' test, without metabolic activation, to test the mutagenicity of arsenic trioxide on 5 (or 4) strains of S. typhimurium. Positive controls (4-nitroquinoline-N-oxide and N-methyl-N'-nitrosoquanidine) were used. The results were negative for arsenic trioxide.

(ii) Bacterial DNA Repair

Nishioka (1975) examined the mutagenic effect of sodium arsenate and sodium arsenite on strains of Bacillus subtilis by rec-assay. For the assay, a 0.05 ml aliquot of either arsenic compound (at a concentration of 0.05 M) was dropped on filter paper which was placed on plates containing B. subtilis strains H_{17} (rec^+, arg^-, trp^-) or M_{45} (rec^-, arg^-, trp^-). Sodium arsenate was a positive mutagen, but its effect was less distinct than the effect with sodium arsenite.

(iii) Chromosomal Aberration Studies

1. In Vitro Cytogenetics

Oppenheim and Fishbein (1965) cultured human peripheral leukocytes stimulated with phytohemagglutinin (stimulates mitotic activity) for 72 hours in 0.5, 1.0, 3.3, and 10.0 uM potassium arsenite solutions (arsenic trioxide in dilute potassium bicarbonate). An additional culture was treated for 48 hours in a solution of 1.0 uM concentration. At a

concentration of 1 uM, potassium arsenite induced chromosomal damage, such as chromosome gaps and breaks, translocations, dicentrics, and ring forms. Potassium arsenite also supressed cell division, increased the number of broken metaphase plates, and increased cell damage at the higher concentrations. These effects increased from the 48-hour to the 72-hour treatment, with the exception of cell division which was suppressed more at 48 hours.

Paton and Allison (1972) investigated the effects of subtoxic doses of sodium arsenite and sodium arsenate on cultured human diploid fibroblasts and leukocytes. In chromosomal studies, leucocyte cultures were treated for 48 hours with 2.9×10^{-9} to 1.8×10^{-8} M solutions of sodium arsenite or 5.8×10^{-9} M sodium arsenate. Of the metaphase plates examined, 60% (sodium arsenite) and 5% (sodium arsenate) of the cells had chromatid breakage. In fibroblast cultures treated for the last 24 hours of culture with 2.9×10^{-9} to 5.8×10^{-8} M sodium arsenite, 20% of the metaphase plates showed chromatid breakage. These values were significantly higher ($p = 0.01$) than the control values for leukocytes (2%) and for fibroblasts (3%).

To study long-term effects, cultured fibroblasts and leukocytes were treated for several weeks with subtoxic doses of these arsenic salts. The cells were often over-contracted but otherwise did not differ from the

control cells. In short-term exposure (24 to 48 hours) of fibroblast and leukocyte cultures to sodium arsenite and sodium arsenate, there was significantly more chromatid breakage than in the control cultures. The breaks were often located close to the end of a chromosome. The incidence of chromatid exchange was higher in leukocytes than in fibroblasts.

2. In Vivo Cytogenetics

Petres et al. (1970; 1977) did chromosomal analyses of lymphocytes from a group of patients previously exposed to arsenic and from a control group with no previous exposure to arsenic. Since the 1977 study appears to include many of the same persons in the first study, only the latter paper will be discussed. The control group consisted of 14 psoriasis patients and 17 healthy patients, both without a history of exposure to arsenic. The test group included 14 psoriasis patients medically treated with arsenic and 17 vineyard workers who had used arsenical pesticides. In some cases the last exposure to arsenic occurred more than 30 years previously. The control group averaged 14 years younger than the exposed patients. The male/female sex ratio was near unity in the control group but was approximately 6/1 for the exposed group. All patients who had been exposed to arsenic had palmar and plantar hyperkeratosis and some history of cutaneous carcinomas.

Several observations were made on cultured lymphocytes:

1. Chromatid breaks, acentric fragments, and dicentric chromosomes occurred more frequently in cells from persons exposed to arsenic (76/1121=0.07) than in control cells (3/1247=0.002) ($p<0.001$). Two dicentric chromosome-containing cells were found in treated persons; none were noted in controls. No information was given as to the distribution of the aberrant cells among the subjects;

2. Aneuploidy occurred significantly more frequently in cells from treated subjects than in cells from controls (26.7 and 7.1% of the treated and control cells, respectively). It is not known if the aberrant cells were distributed randomly among subjects. The high frequency of aneuploid cells in exposed persons is difficult to interpret, because essentially all aberrant cells were hypodiploid; cells with hyperdiploid chromosome numbers were not found; and

3. The incorporation of radioactively-labeled thymidine and uridine into DNA and RNA decreased in a dose-response fashion in cultures of lymphocytes, from control persons, treated with exogenous sodium arsenate (0.05 to 100 ug/ml). The effect of arsenate on nucleotide transport into cells and on thymidine/uridine phosphorylation to nucleotides for incorporation into nucleic acids was not assessed.

3. Dominant Lethal Effects

Using sodium arsenite, Sram and Bencko (1974) performed a dominant lethal test with ICR-SPF mice using both acute and chronic multi-generation dosing regimens, and tested for synergistic effects with TEPA, a positive control mutagen. To determine if sodium arsenite can induce dominant lethal mutations, a toxic dose of 250 mg/kg was applied perorally (acute test). The results were negative.

In a second study of chronic application over four generations, weaned male mice were given either 10 or 100 mg of arsenic per liter of drinking water *ad libitum* over an 8-week period. Based on consumption of water, the actual intake of arsenic was 1.6 mg/kg per day (10 mg/liter)

and 9 to 10 mg/kg per day (100 mg/liter). During each of the eight weeks, several male mice (P_1) were mated, and the next three generations came from these matings. In males given 10 mg/liter, the overall dominant lethality and preimplantation lethality increased in the second and third weeks of mating. In other weeks there was no difference from the controls. The males exposed to 100 mg/liter (subtoxic) showed no change in the frequency of dominant lethal mutations or male fertility.

A third study of synergistic effects with TEPA (reference chemical mutagen) was done. A single intraperitoneal dose of 1 mg of TEPA per kg was given to males of the F_3 generation that were given either 10 or 100 mg/liter of sodium arsenite. For treatment with TEPA and 10 mg/liter of arsenic, there was no increase in the number of mutagenic effects over that seen with 1 mg of TEPA. At 100 mg/liter of sodium arsenite and TEPA, there was an increase in the overall dominant and preimplantation lethality which increased significantly in the second week.

(iv) DNA Damage

Burgdorf et al. (1977) found an elevated sister chromatid exchange rate (SCE) in lymphocytes from six patients who had used Fowler's solution (containing potassium arsenite) compared to lymphocytes from 44 unexposed controls. The lymphocytes were cultured in total darkness with phytohemagglutinin, penicillin-streptomycin, and bromodeoxy uridine and treated with colchicine for three hours. The cells were fixed and stained for SCE. Centromeric exchanges were differentially included in the total SCE counts.

The arsenic-exposed patients had a mean of 14.00 SCE/mitosis with a range of 4 to 30 (standard deviation, 3.12). The controls had a mean of 5.8 SCE/mitosis with a range of 1 to 23 (standard deviation, 2.27). There was no difference in chromosomal breakage between the two groups.

(v) DNA Repair Studies

Rossman et al. (1975) irradiated strains of *E. coli*, differing only in repair markers, with UV light and exposed the cells to various concentrations of sodium arsenite. Culture samples were irradiated in petri dishes with a 15 W germicidal lamp at a distance of 50 cm. After exposure, serial dilutions were made and plated onto nutrient agar containing the following concentrations of sodium arsenite: 10^{-5}, 10^{-4}, 10^{-3}, and 10^{-2} M.

The wild type (WP2) and excision-repair deficient strains WP6 and WWP2 (polA$^-$ and uvrA$^-$, respectively) showed decreased survival in the presence of 0.1 mM of sodium arsenite. The magnitude of the effect increased with increasing UV doses. There was no apparent effect on survival of the unirradiated cultures of WP2, but there was a loss of colony-forming ability at a concentration of 5 mM. Sodium arsenite had no effect on the survival of WP10 cells (recA$^-$, deficient in post-replication repair) at any dose. The survival of WP5 cells (lex$^-$) was increased significantly (statistical significance not given) by sodium arsenite, and the effect decreased with increasing UV doses. The authors postulated that arsenite inhibits a recA$^-$ dependent step in DNA repair. For increased survival of the lex$^-$ mutant, WP5, it is suggested that in the absence of the lex$^+$ gene, the arsenite-sensitive recA-dependent function is deleterious.

Rossman et al. (1977), based on the findings in their first paper, attempt to support their hypothesis that arsenicals inhibit some aspect of DNA repair, specifically that sodium arsenite affects post-replication repair.

Strains of *E. coli* differing only in repair markers were irradiated with UV light and then plated onto medium containing 0.1 mM sodium arsenite. The strains used were:

WP2 (wild type for repair); WP6 and WWP2, both excision-repair deficient; WP10, deficient in post-replication repair; and WP5. Compared to control cultures, sodium arsenite decreased the survival rate of UV-exposed cells of strains WP2, WWP2, and WP6. There was no effect on survival of strain WP10, and survival increased in strain WP5. Sodium arsenite also had no effect on cultures that were not irradiated.

The excision-repair deficient strain WWP2 is more subject to mutation than the wild type WP2. The mutation frequency for this strain was measured after exposure to UV light, by scoring for trp^+ revertants. Cultures were exposed to UV light and plated with 1 mM sodium arsenite. Controls at each UV dose level were plated onto plain nutrient agar. Sodium arsenite decreased the absolute mutation frequency and the number of mutations per survivor at all UV exposures. At higher UV levels, survival of the cells dropped below 1%.

Based on their data, the authors speculated that sodium arsenite inhibits post-replication repair of DNA by inhibiting a recA-dependent function such as SOS repair. This DNA repair pathway is inducible in damaged cells (i.e., by UV exposure), repressed in undamaged cells, and is error-prone. After UV exposure, sodium arsenite decreased mutagenesis in strains WP2, WWP2, and WP6 containing the $recA^+$ and lex^+ genes (upon which mutagenesis depends).

The effect of sodium arsenite (inhibition of post-replication repair) on the survival of UV-irradiated strains of E. coli increased with increasing irradiation. The authors believe this supports their hypothesis that post-replication repair is inhibited by sodium arsenite. This becomes more important at higher UV doses due to saturation of the excision repair capacity.

When Jung et al. (1969) exposed human epidermal cells to sodium arsenate, there was a temporary reduction in enzymatic dark repair activity of DNA. Dark repair is the non-light dependent cell repair mechanism consisting of several enzymatic steps by which single-strand DNA lesions can be recognized, excised, and repaired.

Skin grafts from human male subjects were incubated for either one or four hours in medium containing either 10^{-5} or 10^{-6} M sodium arsenate. The cultures were then irradiated with an unfiltered xenon lamp and incubated with ^{3}H-methyl thymidine for four hours. In the stratum basale, stratum spinosum, and stratum granulosum, the thymidine labeling was visible in many nuclei as "light marking", which is regarded as repair marking and is dependent on the intensity of irradiation. A statistical evaluation showed that repair activity fell significantly ($p<0.05$) as a

result of incubation with sodium arsenate. There was no statistically confirmed difference between the incubation periods of one and four hours or between arsenic concentrations of 10^{-5} and 10^{-6} M.

Jung and Trachsel (1970) showed that sodium arsenate inhibited DNA synthesis and DNA dark repair in human epidermal cells. The cells were cultured in medium containing 10^{-5} or 10^{-6} M sodium arsenate for one or four hours. The cultures were then radiated with an unfiltered xenon lamp. Radio-labeled thymidine was added to the cells which were then incubated for four hours. The cells were fixed, stained, and autoradiographed. The number of premitotic "marked" cells per 1,000 cells was calculated and used as the mitotic index.

Cells from various stages of the incubation process were examined: control cells, cells irradiated only, cells incubated with sodium arsenate (1 hour), and cells irradiated and cultured with sodium arsenate. Radiation and arsenic alone reduced the mitotic index, but this effect was significantly greater with simultaneous exposure ($\alpha = 0.01$).

To measure the effectiveness of the dark repair system, the number of silver grains per cell surface was measured for 200 epidermal cells (measure of thymidine incorporation into DNA). There was a statistically significant ($\alpha = 0.05$) reduction in dark repair due to

sodium arsenate. There was no difference between a 1- or 4-hour incubation time or between 10^{-5} and 10^{-6} M concentrations of arsenate.

The Agency concludes that there is multi-test evidence for the mutagenicity of inorganic arsenates and arsenites. Based on Nishioka (1975), there is evidence that both arsenate and arsenite compounds are positive point mutagens. In studies by Jung et al. (1969) and Rossman et al. (1977), inorganic arsenate and arsenite inhibited DNA repair mechanisms. Both in vitro and in vivo studies show that arsenite and arsenate induced chromosomal aberrations in various cultured human cells and in lymphocytes taken from psoriasis patients and vintners.

The Agency concludes, therefore, that sufficient data exist on the mutagenicity of both trivalent inorganic arsenic (arsenite) and pentavalent inorganic arsenic (arsenate). Section I.J presents strong evidence of the ability of inorganic arsenic to interconvert in vivo (arsenite to arsenate, and arsenate to arsenite). As discussed in Section III.A, this interconversion makes trivalent arsenic a metabolite of pentavalent arsenic, and pentavalent arsenic a metabolite of trivalent arsenic.

Therefore, in the case of pentavalent inorganic arsenic, the Agency stresses that the RPAR is being issued for two reasons: 1) that pentavalent arsenic is a mutagen

and 2) that pentavalent arsenic is reduced in vivo to trivalent arsenic, which also acts as a mutagen. The EPA concludes that either characteristic is a sufficient criterion for the issuance of an RPAR.

The basis for issuance of an RPAR for trivalent inorganic arsenic is similar: 1) that trivalent arsenic is a mutagen and 2) that trivalent arsenic is oxidized in vivo to pentavalent arsenic, which also acts as a mutagen.

(b) Studies on Which Further Information is Sought: Metabolic and Cellular Toxicity Effects

This section addresses effects of inorganic arsenicals on genetic material and processes that are probably due to metabolic and cellular toxicity, but for which insufficient evidence exists to initiate a rebuttable presumption. The Agency requests registrants or other interested persons, having information on these effects, to submit such data to the Agency. The following studies indicate that inorganic arsenicals affect the mitotic process and induce chromosomal pulverization as well as inhibition of the incorporation of important precursors into DNA and RNA.

Skipper et al. (1951) injected CFW strain mice intraperitoneally with an 18.0 mg/kg dose of potassium arsenite. This was immediately followed by an injection of ^{14}C-sodium formate. The control mice received only the injection

of labeled sodium formate. After six hours, all groups of mice were sacrificed. Potassium arsenite markedly reduced the incorporation of ^{14}C-formate into nucleic acid purines. In the rat, formate carbon is a required precursor of the bases in deoxyribonucleic acid (guanine, adenine, and thymidine) and of ribonucleic acid (guanine and adenine).

Sawada and Rebhun (1969) studied the effects of sodium arsenite on eggs of the annelid worm Pectinaria gouldi which develop the first mitotic apparatus (MA) after they are shed into sea water. The MA will remain at metaphase for several hours until the egg is fertilized. Prior to the formation of the MA, eggs were immersed in solutions of sodium arsenite in sea water (concentration not specified). This completely inhibited the formation of the MA in annelid worm eggs. If development of the MA was allowed to occur, the minimum concentration for its dispersion was 5×10^{-3} M This dispersion was irreversible.

Petres and Hundeiker (1968) cultured phytohemagglutinin-stimulated human lymphocytes of peripheral blood from nonarsenic-exposed donors for 72 hours. At either 24 or 48 hours before terminating the cultures, sodium arsenate was added at the concentrations of 10^{-6} and 5×10^{-6} g/ml of culture media. Control cultures were prepared without sodium arsenate. Compared to the controls, the

number of mitoses in the cells treated with sodium arsenate was reduced to approximately one fifth (1/5) the expected number. Chromosome pulverization occurred consistently in cultures incubated for 24 or 48 hours with sodium arsenate.

Petres and Berger (1972) studied the potential cytotoxic and mutagenic effects of sodium arsenate tested in vitro on cultured human lymphocyte cells of peripheral venous blood from non-arsenic-exposed donors. The cells were cultured for 72 hours in a medium containing phytohemagglutinin. Forty-eight hours before termination of the cultures, sodium arsenate was added at the following concentrations: 3×10^{-5}, 1×10^{-5}, 5×10^{-6}, 2×10^{-6}, 1×10^{-6}, 5×10^{-7}, 1×10^{-7}, and 5×10^{-8} g/ml of culture medium. Control cultures were grown without sodium arsenate. ^{3}H-thymidine was also added to both test and control cultures. The mitotic index decreased considerably only with the higher sodium arsenate solutions. Serious nuclear division disorders and metaphase plate pulverization occurred at low doses. At the concentrations of 1×10^{-7} and 2×10^{-6} g/ml, there were 36.3% and 80 to 100% pulverizations, respectively. The ^{3}H-thymidine labeling index was significantly reduced at the mid-level doses compared to the control values. This reduction in the index indicates inhibition of ^{3}H-thymidine incorporation into DNA.

Petres et al. (1974) added sodium arsenate to cultures of phytohemagglutinin-stimulated human lymphocytes of peripheral venous blood from non-arsenic-exposed donors. The incorporation of radioactively labeled thymidine and uridine into DNA and RNA was measured by scintillation counting and autoradiography. Sodium arsenate was added 24 hours before termination of the 72-hour incubation period at the following concentrations (ug per ml of medium): 0.05, 0.1, 0.5 (thymidine only), 1.0, 2.0 (thymidine only), 5.0, 10.0, 30.0, 50.0, and 100.0. Control cultures were simultaneously prepared without sodium arsenate. Six hours before termination of the cultures, either H-^{3}thymidine or H-^{3} uridine was added to one set of the cultures.

For scintillation counting, cultures were prepared with sodium arsenate concentrations of 0.05, 0.1, 10.0, 30.0, 50.0, and 100.0 ug of sodium arsenate per ml of culture medium. Controls without sodium arsenate were also prepared. Eighteen hours before termination of the 72-hour culture period, either ^{14}C-thymidine or ^{14}C-uridine was added to a culture.

The marker index obtained using autoradiography indicated that inhibition of ^{3}H-thymidine incorporation into DNA began at low concentrations of sodium arsenate (i.e., 0.1 ug/ml). This inhibition increased with increasing

concentrations, and at 100 ug/ml there was 97% inhibition compared to controls. Inhibition of ^{3}H-uridine incorporation into DNA began at concentrations of sodium arsenate greater than 10 ug/ml. At 100 ug/ml there was 50% inhibition compared to controls.

Using scintillation counting, inhibition of ^{14}C-thymidine incorporation into DNA was detected at 1.0 ug/ml and increased with higher concentrations. Inhibition of ^{14}C-uridine incorporation into RNA was detected at concentrations greater than 10.0 ug/ml.

Petres et al. (1975) cultured phytohemagglutinin-stimulated human lymphoctes of peripheral blood from non-arsenic-exposed donors for 72 hours. From 1 to 18 hours before termination of the cultures, either 10, 30, or 50 ug of sodium arsenate was added per ml of culture medium. Parallel control cultures were prepared without sodium arsenate. Later ^{14}C-thymidine was added to each culture. In human lymphocyte cultures exposed to various levels of sodium arsenate for one hour, there was a 17% increase in the incorporation of ^{14}C-thymidine into DNA. As the exposure time increased, there was a general decrease in the incorporation rate. After 18 hours of incubation with 10 ug of sodium arsenate, the incorporation rate was inhibited up to 35%. At the higher concentrations, there was 65 to 70% inhibition.

Baron et al. (1975) exposed phytohemagglutinin-stimulated human lymphoctes to various concentrations of sodium arsenate, ranging from 0.05 to 100 ug/ml of culture medium, for 24 hours. Radioactively labeled triphosphates and amino acids were also added to the cultures. The incorporation of ^{14}C-thymidine into DNA was inhibited at a lower dose than the incorporation of ^{14}C-uridine into RNA. This confirmed the previous study by Petres et al. (1974). The incorporation of ^{14}C-TTP into DNA was inhibited to the same extent as ^{14}C-thymidine. ^{14}C-UTP incorporation into RNA was similar to that of ^{14}C-uridine only at levels below 1.0 ug of sodium arsenate per ml of culture medium. At higher doses, the incorporation of ^{14}C-UTP was inhibited less than that of ^{14}C-uridine.

The inhibition of incorporation of ^{14}C-ATP with increasing sodium arsenate doses was parallel to the incorporation of ^{14}C-UTP and ^{14}C-TTP up to a concentration of 10 ug/ml. Above this concentration, the incorporation of labeled ^{14}ATP into nucleic acids was greater than ^{14}C-TTP and less than ^{14}C-UTP.

A decrease in the incorporation of ^{14}C-alanine and ^{14}C-leucine into cellular protein was not found below a sodium arsenate concentration of 10 ug/ml. Even at the higher doses, there was less inhibition of incorporation than that of the labeled triphosphates and nucleosides.

Tsuda (1974) exposed L strain mouse fibroblast cells to three concentrations of sodium arsenate (0.5×10^{-4} M, 10^{-4} M, and 0.33×10^{-3} M) from 1 to 48 hours. The effects on each phase of mitosis were determined by changes in the mitotic index (# mitoses per 100 cells). As the concentration of sodium arsenate increased, the indices for prophase, metaphase, and mitosis increased compared to the control values. The telophase index decreased, while the index for anaphase remained in the control range of values. Complete recovery of cells exposed to sodium arsenate for one hour appeared to be delayed for approximately six hours. There was also no apparent increase in the mitotic index over the control index.

(3) Reproductive and Fetotoxic Effects in Mammalian and Avian Species

40 CFR 162.11(a)(3)(ii)(B) provides that a rebuttable presumption shall arise if a pesticide "(p)roduces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety." This section reflects concern that chronic exposure to chemicals may result, among other things, in injury to the reproductive system and/or the fetus and provides that a rebuttable presumption shall arise if chronic chemical exposure in test animals produces such results.

Studies discussed in this report (and summarized in Table 24) show that both trivalent (arsenite) and pentavalent (arsenate) forms of inorganic arsenic can act as teratogenic agents. The Agency concludes that, without exception, a rebuttable presumption against registration exists for all inorganic arsenical pesticides based on the teratogenic effects criteria. This decision takes into account anticipated exposure to inorganic arsenical pesticides as discussed in Section III.

Beaudoin (1974) gave single intraperitoneal injections of sodium arsenate in distilled water to pregnant Wistar rats on one of days 7 through 12 of gestation. The dosage level of sodium arsenate ranged from 20 to 50 mg/kg of body weight. The embryolethal and teratogenic effects of sodium arsenate were dose-dependent and peaked on days 9 and 10 of treatment. Resorption of fetuses was day- and dose-dependent. The percentage of resorbed fetuses was the highest at 40 mg/kg and peaked for treatment on day 9. Fetal weights for days 8, 9, and 11 of treatment were significantly less ($p < 0.01$) than the controls.

The frequency of malformed fetuses markedly increased when the dosage went from 20 to 30 mg/kg. Little additional fetal malformations occurred with 40 mg/kg. The greatest degree of fetal abnormality occurred on day 9 of gestation. A dose of 50 mg/kg of sodium arsenate was lethal to embryos for all days (7 through 12) of treatment. Teratogenic effects were not observed in fetuses treated with 20 or 30

Table 24. Summary of Studies on Inorganic Arsenic Teratogenicity in Animals.

Authors	Chemical(s)	Test Animal	Treatment: Gestation Day(s) Treated / Day(s) Sacrificed		Dosage (mg/kg)	Effects
Beaudoin 1974	Sodium arsenate	Wistar rat	ip	7 - 12 / 20	20 - 50 single dose	Fetal resorption and malformation were dose-dependent and peaked on treatment days 9 and 10. 50 mg/kg was lethal to all embryos. There were no teratogenic effects on days 7 and 11 and few on day 12. Malformations: eye defects, exencephaly, renal agenesis, rib and vertebral defects.
Burk and Beaudoin 1977	Sodium arsenate	Wistar rat	ip	9 - 11 / 20	30 - 50 single dose	Fetal resorption and malformation were greatest on day 9. Renal agenesis occurred mainly from treatment on day 10. Skeletal defects occurred mainly from treatment on days 9 and 10. Fetal weights declined significantly with treatment. Malformations: exencephaly, eye defects, facial clefts, agnathia, hydrocephalus, encephalomeningocele, diaphragmatic hernia.
			ip	10 / 11 - 16	45 single dose	Renal agenesis resulted mainly from growth retardation of the mesonephric duct.
Ferm and Carpenter 1968	Sodium arsenate	golden hamster	iv	8 / 12 - 14	5,20,40 single dose	No malformations at 5 mg/kg. All fetuses killed *in utero* at 40 mg/kg. At 20 mg/kg, there were exencephalic lesions, bulging eyes, and protruding tongue and mandibles.
Holmberg and Ferm 1969	Sodium arsenate	golden hamster	iv	8 / 13	20 single dose	There was fetal resorption and malformation: exencephaly, encephalocele, cleft palate and lip, anophthalmia, ear malformations.
Ferm et al. 1971	Sodium arsenate	golden hamster	iv	8 / 15	15,17.5, 20,25 single dose	Embryo resorption and malformation were dose-dependent. Malformations: exencephaly, rib defects, genitourinary defects, anencephaly, cleft lip and palate, anophthalmia, adrenal agenesis, prognathia. Genital abnormalities were greatest in females, and cleft lip, palate, and rib abnormalities were seen primarily in males.
Hood 1972	Sodium arsenite	albino Swiss-Webster mouse (CD-1 strain)	ip (*in utero*)	7 - 12 / 18	10,12 single dose	There was a high degree of resorption and reduced fetal weights that were day and dose-dependent. Malformations were highest on days 8 to 10: exencephaly, micrognathia, open eye, defective tail, skeletal defects.
Hood and Bishop 1972	Sodium arsenate	albino Swiss-Webster mouse (CD-1 strain)	ip	6 - 12 / 18	25,45 single dose	At 25 mg/kg, there was no significant difference from the controls. At 45 mg/kg, there was increased resorption, especially from days 11 and 12. Fetal weights were reduced especially from days 7 and 9. Fetal malformations were highest on days 7 to 11: exencephaly, shortened jaw, protruding tongue, anophthalmia exophthalmos, open eye, umbilical hernia, tail defects, hydrocephalus, and skeletal defects.
Hood and Pike 1977	Sodium arsenate	albino Swiss-Webster mouse (CD-1 strain)	ip	9 / 18	40 single dose	There was a significant increase in fetal mortality, resorptions, and malformations. There was a decrease in fetal weights due to treatment.
Hood et al. 1977	Sodium arsenate	albino Swiss-Webster mouse (CD-1 strain)	ip	9 - 10 / 18	40	There was less effect on fetal mortality, weight, and malformation in orally treated mice than in those injected.
			oral (p.o.)		120 single dose	
Hood et al. (undated)	Sodium arsenate	albino Swiss-Webster mouse (CD-1 strain)	ip	7 - 15 / 18	40 single dose	There was a significant decrease of fetal weights from treatment on days 7 or 10. There was a significant increase in fetal resportion and death. (Prenatal mortality was higher than in the orally treated mice.) Treatment on days 9 or 10 resulted in increased malformations such as exencephaly, shortened jaw, open eyes, kinked tail, and skeletal defects.
			oral (p.o.)	7 - 15 / 18	120 single dose	There was a significant reduction of fetal weights from treatment on days 10, 11, or 15. The number of dead or resorbed fetuses increased significantly from day 11 treatment. Oral treatment did not increase significantly the incidence of gross malformation, but a trend was evident. Malformations: oligodactyly, micromelia, kinked tail. Skeletal defects increased significantly only from treatment on day 9 (same as with ip treatment).
Matsumoto et al. 1974	Sodium arsenite	mouse (ICR strain)	oral (p.o.)	9 - 11 / 18	10, 20 40 daily dose	There were 5.7% (10 mg/kg), 0% (20 mg/kg), and 4.2% (40 mg/kg) fetal malformations compared to 0% for controls (no statistical values given). Malformations were bent tail, cleft palate, open eyelid, and bone deformities (40 mg/kg). There was reduced fetal growth and ossification of cervical vertebrae and leg/tail bones.
			ip	6 - 11 / 18	5,10, 20 daily dose	There were 3.5% (5 mg/kg) and 1.3% (10 mg/kg) fetal malformations compared to 1.2% for controls (no statistical values given). Malformations were open eyelid, bent tail, club foot, and kidney deformity (10 mg/kg). As in oral treatment there was reduced growth and ossification.

Franke et al. 1936	Sodium arsenite	chicken embryo	injection	not specified	0.01 - 1.0 ppm single dose(1)	There were only ectopic abnormalities (unspecified) and these occurred at the lowest dosage. There were no abnormalities at 1.0 ppm.
Ridgway and Karnofsky 1952	Potassium arsenite, Arsenic pentoxide, Sodium arsenate	Leghorn chicken embryos	injection	4, 8 18	0.2 mg/egg single dose(2)	Embryos injected on the fourth day with sodium arsenate were stunted with mild micromelia and abdominal edema. There were no effects with potassium arsenite and arsenic pentoxide. There were no effects in the eight-day old embryos.
Puzanova and Doskocil 1976	Pentavalent arsenic	Leghorn chicken embryos	injection	2 - 4 varied	3ug/egg single dose(3)	Embryos treated day 2 of incubation were normally differentiated. Of embryos (19) surviving day 3 treatment, 21% had abnormalities (deformed beak, lumbar lordosis, body distortion, wrinkled brain vesicles, incomplete body walls). Thirteen (13) embryos survived day 4 treatment; 32.5% had abnormalities such as impaired mesonephros, kyphosis of body, hyperlordosis and small amnion. Compression of embryos in deformed amnion may have caused defects.
Schroeder and Mitchner 1971	Soluble arsenite salt	Charles River mouse	oral (drinking water)	through 3 generations	5 ppm ad libitum(4)	Arsenic increased the ratio of males to females and reduced the litter size.
Kimmel and Fowler (letter 1977b)	Sodium arsenate Sodium arsenite	rat	oral (drinking water)	1 - 21 21	30,90 ppm ad libitum(5)	No adverse effects.
James et al. 1966	Potassium arsenate	Yearling ewe	oral (capsule)	treated for 18, 45, 140 and 147 days	0.5 - 0.75	All lambs were normal. One lamb born to a ewe given 0.75 mg/kg (later reduced to 0.5 mg/kg due to toxicity) was very small.
Morris et al. 1938	Arsenic trioxide	Osborne - Mendel rat	oral (food)	treated from 4 wk. old to 1 yr.	27, 215 mg in diet(6)	There were no significant differences in fertility and fecundity between the experimental and control groups. This was also true of the F_1 generation when treated similarly.
Kojima 1974	Arsenic trioxide	Wistar rat	oral (food)	treated prior to and during gestation	10, 50, 100 ppm in diet(7)	No significant difference in the number of litters between treated and control rats.

1. Total As: 0.01 - 1.0 mg/kg (Franke et al. 1936).
2. Total As: 4 mg/kg (Romanoff and Romanoff 1949).
3. Total As: 0.06 mg/kg (Romanoff and Romanoff 1949).
4. Daily dose for adult females 0.56 - 2.0 mg/kg (Letter 1978).
5. Total As: 2.25-3.75 mg/kg; 6.75-11.25 mg/kg (Letter 1977b).
6. 9.2 mg/kg/day; 79 mg/kg/day (Morris et al. 1938).
7. Total As: 15.4 mg/kg; 77.1 mg/kg; 154.3 mg/kg (Kojima 1974).

mg/kg on days 7 and 11. On day 12 there were some skeletal defects. Fetal malformations included eye defects, exencephaly, renal agenesis, and rib and vertebral defects. Atlas ossification centers were absent or rudimentary in 63% of the fetuses from dams treated on day 9 of gestation.

Burk and Beaudoin (1977) found that there was a high incidence of malformations (including renal agenesis) and resorptions, both of which were dose-dependent and related to the time of treatment, in embryos from Wistar rats which had been injected intraperitoneally with sodium arsenate.

In a preliminary study, pregnant rats were given a single intraperitoneal injection of aqueous sodium arsenate (30 to 50 mg/kg of body weight) on one of days 9 through 11 of gestation. The rats were sacrificed on day 20. The number of fetal resorptions and malformed survivors (soft tissue and skeletal defects) increased with increasing concentrations of sodium arsenate for each day. The percentages of both were greatest on day 9 and decreased thereafter. Malformations that occurred primarily on day 9 of gestation were exencephaly, eye defects, and facial clefts. There were also cases of agnathia, hydrocephalus, gastroschisis, encephalomeningocele, and diaphragmatic hernia. Day-10 treatment resulted primarily in urogenital system defects. Skeletal malformations occurred primarily on days 9 and 10. Fetal weights declined as the concentration of sodium arsenate increased.

In the second experiment to study renal agenesis, the pregnant rats were injected with 45 mg/kg on day 10 of gestation. This was the most effective dose for production of renal agenesis, with minimal embryonic death. The rats were sacrificed on days 11 through 16. The first abnormal fetuses (from day-12 treatment) showed growth retardation of the mesonephric duct. For treatment days 13 through 16, retardation of the duct eventually prevented formation of the ureteric bud and resulted in renal agenesis. In some cases the mesonephric duct failed to connect with the cloaca, and there was degeneration of the metanephric blastema. Gonadal agenesis was not observed in any of the embryos from treated rats, but in some cases there was incomplete descent of the testes.

Fairm and Carpenter (1968) intravenously injected either 5, 20, or 40 mg/kg of sodium arsenate in distilled water into the lingual vein of pregnant golden hamsters (Cricetus auratus) on the eighth day of gestation. Control animals received only distilled water. The hamsters were sacrificed on days 12 through 14. All embryos died in utero at 40 mg/kg of sodium arsenate. Exencephalic lesions were induced at 20 mg/kg of body weight, and no malformations occurred with 5 mg/kg. The exencephalic lesions were markedly consistent and specific, and often within a litter a majority of the survivors showed strikingly similar defects. Other effects included bulging eyeballs and protruding tongue and mandibles. Histological examination of the placental membranes showed no abnormalities.

Holmberg and Ferm (1969) injected a sublethal dose (20 mg/kg) of sodium arsenate into the lingual vein of pregnant golden hamsters (Cricetus auratus) on the eighth day of gestation. After sacrifice on the thirteenth day, the embryos exhibited several types of malformations, and the most prominent of these in all litters was exencephaly. Other abnormalities included encephalocele, cleft palate and lip, unilateral and bilateral microanophthalmia, and ear malformations. Sodium arsenate induced a total of 49% malformed embryos and 35% resorbed embryos.

Ferm et al. (1971) injected sodium arsenate intravenously into pregnant golden hamsters (Cricetus auratus) at three different times on the eighth day of gestation. The three groups were treated with sodium arsenate at 9 AM, 3 PM, and 9 PM (6-hour intervals) at the following doses (mg/kg): 15, 17.5, 20, and 25 (9 PM only). Control hamsters were injected intravenously with similar volumes of demineralized water. The females were sacrificed on the morning of the 15th day of gestation.

Within each of the three periods of treatment, the rate of embryo resorption and malformation increased with increasing doses of sodium arsenate. A comparison of the three groups showed that the resorption rate decreased the later the time of treatment on the eighth day of gestation.

The rate of malformation did not follow a similar pattern except at 15 mg/kg sodium arsenate. Malformations were slightly more frequent in the 9 PM group at the higher doses. A marked increased in rib malformations accounted for this high malformation rate in the 9 PM period. The overall malformation rate remained constant for all three periods of treatment. Fetal malformations that occurred most frequently included exencephaly, rib malformations, genito-urinary abnormalities, and anencephaly. Other effects seen less frequently were cleft lip and palate, anophthalmia, adrenal agenesis, and prognathic embryos. Cleft lip and palate and rib abnormalities were significantly more frequent in males, and genital abnormalities occurred more frequently in female embryos. The sex ratio for the total malformation rate was close to 1.0.

Hood (1972) gave pregnant albino Swiss-Webster mice single intraperitoneal injections of either 10 or 12 mg of sodium arsenite in distilled water per kg of body weight on one of days 7 through 12 of gestation. Controls were injected with an equivalent volume of distilled water. All mice were sacrificed on the 18th day of gestation. Treatment of the pregnant mice with sodium arsenite resulted in a significant ($p < 0.05$) increase in fetal deaths for all

days 7 through 12 at both dosage levels studied. Most deaths apparently occurred shortly after treatment as determined by the degree of resorption. There was a significant ($p < 0.05$) decrease in fetal weights that was day- and dose-dependent. The higher doses tended to induce a greater effect. The period of greatest susceptibility to teratogenic effects was gestation days 8 through 10. On these days there was a significant ($p < 0.05$) difference from the control value. The most common malformations were exencephaly, micrognathia, open eye, and bent, short, or missing tails. The major skeletal defects were fusion of the ribs and fused or missing vertebral ossification centers. These malformations were found only in fetuses from the groups treated on days 8 through 10 (rib defects) and on days 9 to 10 (vertebral anomalies).

Hood and Bishop (1972) gave single intraperitoneal injections of 25 or 45 mg of sodium arsenate per kg of body weight to pregnant albino Swiss-Webster mice on one of days 6 to 12 of gestation. Controls received an equivalent dose of distilled water. The mice were sacrificed on the eighteenth day of gestation.

In females injected with 25 mg/kg of sodium arsenate, the rates of fetal resorption and malformation were not significantly different from those found in the controls.

Intraperitoneal injections of 45 mg/kg, particularly on treatment days 11 and 12, consistently resulted in significantly increased fetal resorption. Treatment on days 6 through 11 resulted in decreased fetal weights compared to controls ($p < 0.05$). Reduction in fetal weights was the most severe on treatment days 7 and 9. Sodium arsenate induced several fetal malformations that were somewhat day-dependent. Among these were exencephaly, shortened jaw, protruding tongue, anophthalmia, exophthalmos, open eye, umbilical hernia, shortened tail, twisted tail, hydrocephalus, fusion of ribs, forking of ribs, and fusion of vertebrae. The percentage of grossly malformed fetuses, resulting from treatment on the sixth day of gestation, was very low compared to that for days 7 to 11. Treatment on day 12 resulted in the resorption of most fetuses, but those that survived were morphologically normal.

Hood and Pike (1972) found that intraperitoneal injections of sodium arsenate in Swiss-Webster mice on the ninth day of gestation resulted in a significant increase in fetal mortality and malformation and decrease in fetal weight.

Treatment of pregnant mice consisted of single intraperitoneal injections of 40 mg of sodium arsenate in distilled water per kg of body weight. Control mice were injected with water or were not treated at all. All females were sacrificed on the eighteenth day of gestation. The fetuses from treated females had a high rate of resorption and death, gross malformations and skeletal defects, and had greatly reduced fetal weights compared to the fetuses of the controls.

Preliminary work in mice by Hood et al. (1977) showed that the intraperitoneal administration of sodium arsenate had a greater adverse effect on prenatal mortality, fetal weights, and morphological development than oral administration. On gestation days 9 and 10, pregnant albino Swiss-Webster mice (CD-1 strain) were treated with sodium arsenate in distilled water either intraperitoneally (40 mg/kg of body weight) or orally (120 mg/kg). Controls were treated with distilled water. The animals were sacrificed on gestation day 18. The maternal death rate was similar for both treatments, indicating similar levels of toxicity to the females. A dose of 120 mg/kg of sodium arsenate orally administered was required to obtain adverse prenatal developmental effects. There was a much lower level of fetal malformation in the orally treated mice than in those injected intraperitoneally. The intraperitoneal treatment resulted in decreased fetal weights, compared to the controls.

This effect was seen in the oral treatment only when given on day 10 of gestation.

Hood et al. (Undated) administered sodium arsenate orally to pregnant albino mice (CD-1 strain) on one of gestation days 7 through 15. Sodium arsenate was also administered intraperitoneally to other pregnant mice as a positive control. Preliminary studies with oral doses of 40, 50, 60, 80, and 100 mg/kg of sodium arsenate showed no significant differences in effects from the negative controls (water only). Sodium arsenate was then either orally (120 mg/kg) or intraperitoneally (40 mg/kg) administered to pregnant mice on the same series of days.

Compared to the negative controls, dead and resorbed fetuses from orally treated mice increased significantly only on day 11 ($p \leq 0.01$). In the positive control group, however, treatment on days 7, 8, 9, and 11 ($p \leq 0.05$) and 10, 13, 14, and 15 ($p \leq 0.01$) resulted in a significant increase in the number of dead and resorbed fetuses compared to the negative controls. The positive controls (ip) also had a higher prenatal mortality than the groups treated orally on days 7, 11, or 13 ($p \leq 0.01$) and 14 ($p \leq 0.05$).

The incidence of gross malformations from oral treatment did not increase significantly over the negative control values, although the results were suggestive of such an effect. However, intraperitoneal treatment

on days 9 ($p \leq 0.01$) and 10 ($p \leq 0.05$) resulted in an increase in malformations over the negative control values. The gross malformations that occurred most frequently with oral treatment were oligodactyly, micromelia, and kinked tail. Those associated with ip treatment were shortened jaw, exencephaly, open eyes, and kinked tail.

Skeletal malformations resulted from either oral or intraperitoneal treatment on day 9 and were significantly increased ($p \leq 0.05$) over the negative control values. Fewer defective fetuses were seen in litters from treatment on other days. The most common defect was fused ribs with some cases of fused vertebrae. There was no significant incidence of visceral defects.

Compared to the negative controls (water only), oral treatment resulted in significantly lower fetal weights on treatment days 10 and 11 ($p \leq 0.01$) and 15 ($p \leq 0.05$). Fetuses from females that received sodium arsenate by injection on days 7 or 10 ($p \leq 0.05$) or 9 ($p \leq 0.01$) weighed significantly less than those from females receiving only water.

Matsumoto et al. (1974) studied the effect of sodium arsenite on fetuses of pregnant ICR mice that were given the compound orally or intraperitoneally. One group of mice was given 10 or 20 mg of sodium arsenite/kg (body weight) intraperitoneally (ip) once a day on days 6, 7, and 8

after conception. Another group received 5 or 10 mg/kg (ip) once a day on days 9, 10, and 11 after conception. A third group was given 10, 20, or 40 mg/kg orally once a day on days 9, 10, and 11 after conception.

The percentage of malformed fetuses from ip-treated pregnant females was 3.5% (5 mg/kg) and 1.3% (10 mg/kg) compared to 1.2% for the controls (no statistical values given). The percentage of resorbed or dead fetuses was 7.3% (controls), 12.1% (5 mg/kg), and 45.8% (10 mg/kg). In the group of orally treated mice, the percentage of malformed fetuses was 5.7% (10 mg/kg), 0% (20 mg/kg), and 4.2% (40 mg/kg) compared to 0% for the controls (no statistical values given). The percent of resorbed or dead fetuses was 9.8% (controls), 4.5% (10 mg/kg), 5.5% (20 mg/kg), and 30.4% (40 mg/kg).

The malformations observed with oral treatment were bent tail, cleft palate, open eyelid, and bone deformities (40 mg/kg only). Malformations from ip treatment were open eyelid, bent tail, club foot, and kidney deformity (10 mg/kg only). From both ip and oral treatment there was reduced fetal growth and reduced ossification of cervical vertebrae and leg and tail bones.

Franke et al. (1936) tested the effect of sodium arsenite on the development of chick embryos. Sodium arsenite was injected into the eggs at the following concentrations: 0.01, 0.05, and 0.10 ppm. There were no

dead chicks in the group that received 0.01 ppm, but 6.7% had abnormalities of ectopic form (unspecified by the authors). At 0.05 ppm, 14.3% of the embryos were dead, and 14.3% of the survivors were abnormal. In the group that received 0.1 ppm of sodium arsenite, 83.3% of the embryos were dead. However, there were no abnormalities in the surviving chicks.

Ridgway and Karnofsky (1952) injected four- and eight-day-old white Leghorn chicken eggs with potassium arsenite, arsenic pentoxide, and sodium arsenate. When examined on the eighteenth day of incubation, 50% of the chicken embryos, injected via the yolk sac on the fourth day with 0.2 mg/egg of sodium arsenate, were stunted and showed mild micromelia and abdominal edema. This did not occur in the embryos injected on the eighth day. Potassium arsenite and arsenic pentoxide did not produce these effects.

Puzanova and Doskocil (1969) studied the effects of a pentavalent arsenical on white Leghorn chicken embryos. The embryos were given a single dose of 3 ug of arsenic subgerminally or intraaminally on either day 2, 3, or 4 of incubation. The embryos were collected at various times, then fixed and stained.

All embryos from day 2 treatment (collected on 8th day) survived the arsenic application and were normally differentiated.

Of those treated on day 3, 5 died and 19 survived. The dead embryos (collected 12 to 24 hours after treatment) showed abnormalities in the circulatory system (dilatation of vessels, extended heart, hemorrhaging in head and body) and in the brain vesicles (wrinkling). Of the surviving embryos (collected between incubation days 4 and 9), one had a deformed beak, and three had lordosis of the spinal lumbar region. Lordosis indicates a disturbance of amnion development resulting in compression of the embryo in the amnial pouch. There was also some wrinkling of the brain vesicles, body distortion, and inadequate formation of body walls. The other 15 embryos were differeniated normally but were smaller than the control embryos.

From day 4 exposure, 19 embryos died and 13 survived. The dead embryos were collected 12 to 72 hours after treatment. Twelve (12) had circulatory disorders (renostasis, heart dilatation, hemorrhaging), and others showed hyperlordosis, body distortion, wrinkled brain vesicles, and small amnion. Of the 13 survivors, 9 were normal, and 4 showed abnormalities such as impaired mesonephros, kyphosis of the body, hyperlordosis with scoliosis, and small, tight amnion.

In a study by Schroeder and Mitchener (1971), Charles River CD strain mice were bred in special facilities to avoid contamination from trace elements. At the time of weaning, five pairs of mice were selected randomly, fed special food, and given doubly deionized forest spring water. Drinking water containing 5 ppm of a soluble salt of arsenite (form not specified by author) was continuously available to the mice. The control mice received plain deionized water. The mice were allowed to breed at will for six months. Arsenite at this dose level did not affect the rate of growth or long-term survival and did not cause early mortality. In three successive generations of mice, arsenite given via drinking water resulted primarily in an increased ratio of males to females and in a reduction of the litter size.

The following studies showed no teratogenic effects in laboratory animals exposed to various inorganic arsenicals.

Kimmel and Fowler (Letter 1977b) gave pregnant rats either sodium arsenate or sodium arsenite in their drinking water (ad libitum) from gestation day 1 through 21 at exposure levels of either 30 or 90 ppm. There were no adverse effects on fetal weights, number of fetuses per litter, or number of malformations.

James et al. (1966) fed four pregnant yearling ewes potassium arsenate via gelatin capsule. The first ewe received 0.75 mg/kg daily from the first day of gestation.

However, after 18 days, the animal developed toxicosis, and the treatment was discontinued. A second ewe received 0.50 mg/kg daily for the first 45 days of gestation. The remaining two ewes were given 0.50 mg/kg of the salt for the entire gestation period (140 and 147 days respectively). The first ewe gave birth to "a full-term but hairy lamb" that weighed only 1.6 kg. The three other ewes produced normal lambs. Analysis of bone and soft tissue samples indicated that arsenate did not accumulate in detectable amounts in ewes fed arsenic throughout gestation or in lambs from ewes on the reduced treatments.

Morris et al. (1938) fed Osborne-Mendel rats and their offspring a diet containing either 26.8 or 215 mg of arsenic trioxide (approximately 9.2 and 79 mg/kg per day. respectively). The animals were fed the special diets ad libitum from four weeks of age to approximately one year of age.

The rats (P_1) were divided into two groups, one for each dose level. Each group contained 36 rats: 18 controls (9 of each sex) and 18 experimental rats (9 of each sex). The control animals received a diet that was adequate for growth and reproduction. The experimental groups were fed the same diet with the addition of arsenic trioxide. Both

groups of rats were mated at ages of 90 to 110 days as follows: control female x control male, control female x experimental male, experimental female x control male, experimental female x experimental male. Pairs of rats were placed in cages and given the control diet during the mating period, with the exception of experimental females with experimental males. Males were removed from the cages after females showed a positive smear for sperm.

The second generation (F_1) of rats was taken from the second litter of the P_1 females. These animals were given the same diet as their respective parent of the same sex. The mating patterns used for the P_1 groups were also used for the F_1 groups. Only one litter was obtained from this generation. The same patterns of mating were used again for the F_2 group.

No differences in fertility or fecundity were observed between the various experimental groups of both generations (P_1 and F_1) and the control groups. The average weights of P_1 females and their offspring showed no significant differences from the controls.

Kojima (1974) raised groups of male and female albino Wistar rats (P_1) and their offspring (F_1) on a diet containing arsenic (unspecified form, but measured as As_2O_3).

Different groups received various doses of arsenic (10, 50, and 100 ppm), and for each dose level there were five test groups of rats. Group I rats and their offspring received the control diet. Group II received the control diet; and after delactation, the young received the arsenic diet. Group III rats were fed the experimental diet, but the young were given the control food. Group IV rats received the experimental diet, but after delactation the offspring were given the control food. Group V rats and their offspring were fed only the arsenic diet.

The actual amount of arsenic ingested was determined by total food consumption. The P_1 rats each consumed an average of 270 g of food. The arsenic levels consumed by P_1 rats in Groups III, IV, and V were 2.7, 13.5, and 27 mg (10, 50, and 100 ppm respectively). Prior to delactation, F_1 rats of Groups IV and V ingested 0.5, 2.5, and 5 mg (10, 50, and 100 ppm respectively). After delactation, F_1 rats of Groups II and V ingested 10, 50, and 100 mg (10, 50, and 100 ppm respectively).

There were no differences in the number of litters between P_1 rats fed experimental diets containing arsenic and the control group of rats. The young of females raised on the diet containing 100 ppm of arsenic showed no effects in histopathological tests, breaking strength of bones, and Ca and P content of bones and teeth.

Pregnant mice, orally treated with 120 mg/kg of sodium arsenate (Hood et al. 1977; Undated) or 10 mg/kg sodium arsenite (Matsumoto et al. 1974), gave birth to young exhibiting both gross malformations and skeletal defects. Mice given sodium arsenite at 5 ppm in drinking water showed a reduction in litter size over three generations.

Based on these studies and the discussions in Section IV.A, the Agency concludes that there is sufficient evidence for the teratogenic effects of both trivalent inorganic arsenic (arsenite) and pentavalent inorganic arsenic (arsenate). Also there is evidence for the reproductive effects of trivalent inorganic arsenic.

In order to determine whether a rebuttable presumption should be issued on each inogranic arsenical based on these effects pursuant to Section 162.11(a)(3)(ii)(B), the Agency must determine whether an ample margin of safety exists between the levels of arsenate and arsenite which produce these effects and the levels to which humans can reasonably be anticipated to be exposed.

The levels of arsenate and arsenite which produce teratogenic effects discussed in this section of the position document are tabulated in Table 24. Of particular interest are the studies by Hood et al. (1977; Undated) and Matsumoto et al. (1974) in which arsenite or arsenate were administered orally to mice.

In the study by Matsumoto et al. (1974), arsenite was administered orally at dosages as low as 10 mg/kg. At this dosage several malformations (teratogenic effects) were observed including bent tail, cleft palate, and open eyelid. In addition to these effects the author observed reduced fetal growth, and reduced ossification of cervical vertebrae and leg and tail bones. The author did not determine a dosage level (NEL) at which teratogenic effects were not observed.

In the study by Hood et al. (1977; Undated), arsenate was administered orally to mice at 120 mg/kg. This dosage produced skeletal malformations such as fused ribs and some cases of fuses vertebrae. A dosage of 100 mg/kg (NEL) did not produce significant differences from controls.

In order to determine whether an ample margin of safety exists for each inorganic arsenical, the Agency must determine how much of each a 60-kg woman would be exposed to through oral, dermal, and/or inhalation exposure. These determinations were made in the Exposure Analysis (see Section III), and the results are duplicated here (Table 25).

For purposes of this analysis the margin of safety will be defined as the level (dosage in mg/kg) which produced

no observable effect (NOEL) divided by the anticipated exposure:

$$\text{Margin of Safety} = \frac{\text{NOEL}}{\text{Exposure}}$$

In case of inorganic arsenate the NOEL is 100 mg/kg (Hood Undated). For inorganic arsenite, however, all dosages tested produced teratogenic effects (Matsumoto et al. 1974). Therefore, because the NOEL has not been established for inorganic arsenite the lowest effect level [10 mg/kg (Matsumoto et al. 1974)] will be used in the calculation of the margin of safety for inorganic arsenite. It should be noted that if the NOEL for arsenite is found to be only slightly less than 10 mg/kg, this could represent a significant decrease in the corresponding margin of safety. The margin of safety for each use situation discussed in the exposure analysis has been calculated and can be found in table 25.

Table 25. Summary of the Total Theoretical Exposure To Inorganic Arsenic Resulting From the Use of Inorganic Arsenical Pesticides

Chemical or Use Situation	Total Theoretical Exposure (for a 60-kg Woman in mg/kg)	Margin of Safety
Agricultural Situations		
Sodium Arsenite (trivalent)	5.5	1.8
Arsenic Acid (pentavalent)	2.72	37
Sodium Arsenate (pentavalent)	0.65	154
Copper Acetoarsenite (trivalent)	0.32	31
Calcium Arsenate (pentavalent)	0.17	588
Arsenic Trioxide (trivalent)	0.68	15
Lead Arsenate (pentavalent)	0.66	152
Wood Preservative Situations (Sodium Pyroarsenate (Arsenic Trioxide*, Arsenic Pentoxide, Sodium Arsenate, Arsenic Acid, and Ammonium Arsenite*)		
Large Industrial Situations		
Inhalation Exposure	0.05	2000
Dermal Exposure	0.57 - 9.0	11 - 175
Intermittent Application to Wood During On-Site Construction		
Dermal Exposure to Ammonium Arsenite	1.1	91
Exposure From Handling, Sawing, Fabricating, etc, of Treated Wood Products		
Inhalation Exposure	0.27	370
Dermal Exposure	0.24	417
Exposure To Individuals Living or Working In Dwellings Containing Arsenic-Treated Wood		
Inhalation Exposure	$7X10^{-7}$ - 0.002	$5X10^{4}$ - $1.4X10^{8}$
Dermal Exposure	0.03 - 2.59	39 - 3333

* During the formulation of wood preservatives, all trivalent arsenic is oxidized to pentavalent arsenic; therefore, the NOEL for arsenate (100 mg/kg) was used in calculating the margin of safety for all wood preservatives.

It is generally recognized that humans vary in their susceptibility to a toxicant and that humans may, for a variety of reasons, be more sensitive than laboratory animals. Therefore, the Agency concludes that an ample margin of safety does not exist for any use of the inorganic arsenicals and recommends that a rebuttable presumption be issued against the following: ammonium arsenite, arsenic acid, arsenic pentoxide, arsenic trioxide, calcium arsenate, copper acetoarsenite, lead arsenate (basic and standard), sodium arsenate, sodium arsenite, and sodium pyroarsenate.

V. OTHER RELEVANT ADVERSE EFFECTS

This section addresses certain criteria for determination of unreasonable adverse effects which arsenic may produce, but for which insufficient evidence exists to initiate a rebuttable presumption. The Agency requests registrants or other interested persons, having information on these or any other possible adverse effects of inorganic arsenic, to submit such data to the Agency.

A. Delayed Neurotoxicity

Landau et al. (1977) did a pilot study of male workers in a copper smelter using ores high in arsenic (previously studied by Milham and Strong, and Pinto and McGill). Three groups of men were studied. The first group were workers from an area of high arsenic exposure who had 200 ug/liter of arsenic in urine samples for at least the last five years. The second group were workers from a low arsenic exposure area, and the third group consisted of workers from an aluminum plant (no arsenic exposure) and from non-industrial settings.

The men received a clinical and a neurological examination and completed a personal questionnaire. Samples of their blood, urine, hair, and nails were taken. The neurological examination consisted of sensory and/or motor nerve conduction studies on three nerves: the right ulnar (sensory and motor), the right common peroneal (motor), and the right sural (sensory). Both conduction and amplitude were measured for each nerve using standard techniques with surface electrodes.

Urinary arsenic levels corresponded well with worker exposure to arsenic. Nail and hair analyses also clearly distinguished the test groups. Blood arsenic was determined not to be a useful measurement of arsenic exposure.

Exposure to other metals was also investigated. Copper in the blood and urine did not differentiate the groups, while copper levels in the hair did. Cadmium, nickel, and zinc levels in nail tissue were highest in both of the smelter worker groups. Lead values in urine, blood, nails, and hair were generally highest in the smelter workers.

Neurological findings were based on a clinical examination for neuropathies and an objective electrical measurement. From a preliminary analysis, a dose-response relationship was not clearly evident. There was a weak association between the clinical and the objective electrical measurements of the neurological status of each man. A multivariate analysis was used to further study the association of neurological abnormalities from exposure to arsenic and other metals. There was a relationship between lower ulnar sensory amplitude, lower peroneal conduction velocity, and lower peroneal amplitude at the ankle and knee with higher levels of arsenic in urine, hair, and nails.

After further analysis, the authors concluded that there is a statistically significant relationship between arsenic levels (exposure) in body tissues and

reduced neurological response. The clinical examination showed that a higher percentage of the smelter workers had some form of neuropathy compared to the control group. This was after diabetes, trauma, cigarette and alcohol use, use of drugs, and other relevant factors were taken into account. The authors also discussed the various sources of error in the study such as individual day-to-day variability, proximity of workers' homes to the smelter stack, and the effect of age on the neurological responses tested.

Mizuta et al. (1956) reported on 220 patients of all ages who had been poisoned by contaminated soy sauce, with an average estimated ingestation of roughly 3 mg of arsenic (probably calcium arsenate) daily for two to three weeks. In this group, 85% had facial edema and anorexia; fewer than 10% had exanthemata, desquamation, and hyperpigmentation; and about 20% had peripheral neuropathy. Although the majority of the patients' livers were enlarged, relatively few abnormalities were found in liver-function tests; the histopathologic description of five liver biopsies did not reveal severe degenerative changes. There were no findings suggestive of congestive failure, but

electrocardiograms were abnormal in 16 of 20 patients. The patients' symptoms tended to diminish after five or six days, despite continued intake of arsenic, and neurologic symptoms became prominent as much as two weeks after arsenic ingestion was discontinued, at which time urinary arsenic content remained high. Hair was found to contain arsenic at 2.8 to 13.0 ppm near the root, compared with 0.4 to 2.8 ppm in hair near the root from control patients.

Ohira and Aoyama (1973) organized an epidemiological study on babies that drank arsenic-tainted milk in 1955 in Japan. The number of victims was 12,159, of whom 131 died. The disaster was caused by an arsenic-contaminated milk stabilizer.

A survey was made of 214 people of whom 74 were given medical examinations. A questionnaire collected in 1955 was rereviewed. Comparative studies were done on the victims and their brothers and sisters, and on handicapped children in institutions (born from 1953 to 1955) who drank arsenic-tainted milk, those who drank a noncontaminated powdered milk, and those who were fed only maternal milk. Another study was done on children (born between 1954 and 1955) in a district where good records had been kept of their physical growth and mental development through school. They were divided into the same groups as the above study. Examinations were made of 124 patients in another district.

The exposed group had a statistically significant higher rate of the following problems compared to the two control groups. There was apparently no distinct difference between these groups in their prenatal and postnatal development before ingestion of the contaminated milk. The victims had a lower intelligence quotient than their brothers and sisters. Among mentally retarded children in one district, the victim group had a higher rate of severe retardation (IQ below 50) than children who had not consumed the arsenic-tainted milk. The victims showed growth retardation from the age of one year to school age compared to the other groups. There was a higher prevalence of myopia; abnormal EEG findings; higher DMF rate (number of decayed, missing, or filled teeth); and abnormal X-ray findings in facial shape. Although not statistically significant, there was a higher rate of the following problems: mortality, abnormal ophthalmoscopic findings, growth retardation during the school years, retardation of skeletal growth (including finger size), higher peridontal disease rate, narrow vision range, and abnormal X-ray findings in teeth. The authors concluded that the victims were still suffering from many physical and mental disorders after 17 years.

B. Hazard to Wildlife

(1) Population Reductions in Nontarget Aquatic Organisms

40 CFR Section 162.11(a)(3)(ii)(C) provides that a "rebuttable presumption shall arise if a pesticide's [use]...[c]an reasonably be anticipated to result in significant local, regional, or national population reductions in nontarget organisms...."

Menhinick (1962) listed the following possible adverse effects which may result from introduction of pesticides into various ecosystems: a decrease in the number of species and/or genera, a decrease in number and species of large organisms, a compensating increase in numbers of tolerant species, and a shift in trophic structure in favor of lower trophic levels.

When sodium arsenite was registered for use as an aquatic herbicide, it was recommended that arsenic content in treated fresh water not exceed 0.05 ppm (Goudey 1946); a limit of 10 mg/liter [ppm] of arsenic contamination has been recommended for estuarine waters (Lincer 1975).

Except for use of copper acetoarsenite in mosquito control programs, there are no inorganic arsenicals registered for aquatic uses. Indirect sources of arsenic contamination of fresh and marine/estuarine waters, which may adversely affect aquatic organisms, include drift and runoff from agricultural application of inorganic arsenicals, as well as use of wood (for pilings and docks) treated with inorganic arsenical wood preservatives.

(a) Phytoplankton, Zooplankton, and Invertebrates

Reduction in populations of plankton and aquatic invertebrates can have indirect adverse effects on higher organisms by reducing numbers of oxygen-producing algae and through depletion of food sources (Ball and Hooper 1966).

Surber and Meehan (1931) conducted experiments which showed that 2.0 ppm arsenious oxide [arsenic trioxide] equivalent in a commercial sodium arsenite formulation would not kill important fish food organisms. The most sensitive species were chironomid larvae, mayfly nymphs, and fresh water shrimp; these species showed relatively high mortality rates on exposure to 2.5 to 4 ppm As_2O_3. Other studies, however, have shown that use of sodium arsenite can reduce numbers of fish food

organisms. Lawrence (1958) performed studies which showed that two applications to experimental ponds of 4 ppm As_2O_3 as sodium arsenite one month apart reduced the number of bottom invertebrates an average of 34% as compared to control ponds; two applications of 8 ppm As_2O_3 one month apart reduced bottom invertebrates an average of 45% as compared to controls. An application of 4 ppm As_2O_3 one year later to the ponds which had received two applications of 4 ppm resulted in an average increase of 33% in bottom organisms; application of 4 ppm to ponds which had received two applications of 8 ppm, however, resulted in an average decrease of 20% in bottom organisms. The authors also noted that an application of 4 ppm As_2O_3 as sodium arsenite to experimental ponds had killed all the microcrustacae and greatly reduced the population of rotifers.

Cowell (1965) studied the effects of sodium arsenite on plankton populations in farm ponds. At concentrations of 4 ppm during two successive years, sodium arsenite significantly decreased zooplankton populations from at least one sampling depth in three of four treated ponds during the first year; during the second year, sodium arsenite at 4 ppm significantly reduced zooplankton populations, especially rotifers.

Ball and Hooper (1966) studied the effects of ^{74}As-labelled sodium arsenite on pond ecology. A pond with 1/3 acre surface area and a depth of 2 meters was treated with 8 ppm sodium arsenite. Results of the pond study showed that the treatment destroyed a major portion of the phytoplankton. In addition, the number and volume of bottom invertebrates was reduced; reduction ranged from 57% for dragonflies to 100% for mayflies. The authors concluded that sodium arsenite "persists in the ecosystem at low concentration levels and that it has an adverse effect upon many components of the system. Sodium arsenite kills many organisms important to the economy of the pond and modifies the primary producers to such an extent that production is seriously affected."

Gilderhus (1966) also studied the effects of sodium arsenite, at sublethal concentrations, on bottom invertebrates and plankton (Table 26). Nine concrete ponds, with surface measurements of 0.01 acre, were treated with varying concentrations of sodium arsenite over a 16-week period. Table 26 shows the results of the these treatments on bottom organisms and microorganisms.

Table 26. Effects of Arsenic Treatments on Populations of Invertebrates and Microorganisms

Pool	Total Sodium Arsenite Applied (ppm)	Avg. # Bottom[a/] Organisms per sq ft	Avg. # Micro-[b/] organisms per liter
1	4.0	24.7	3,707
2	1.2	99.0	2,328
3	0.4	68.1	3,194
4	4.8	27.9	417
5	1.6	73.4	2,643
6	0.0	58.9	1,211
7	19.2	6.3	178
8	6.4	21.3	1,754
9	0.64	64.1	1,847

a/ Ten families were represented: Oligochaeta, Nematomorpha, Ephemeroptera, Odonata, Coleoptera, Hemiptera, Chaoborinae, Tendipedidae, Ceratopogonidae, and Tabanidae.
b/ Populations counted were rotifers, Cladocera, copepods (adults and nauplii), and microcrustaceans.

Gilderhus (1966) noted that the average numbers of bottom invertebrates in pools at the four highest treatment levels were less than one-half the average found in the other treated pools and control. With regard to plankton, the two pools with the highest treatment levels had substantially reduced average numbers of microorganisms, particularly rotifers. The author also stated that total numbers of microcrustaceans, which are valuable fish foods, appeared greatly reduced in all pools which received totals of 4 ppm or more.

(b) Fish

The PERS report (EPA 1977) on inorganic arsenic (see Section I.H) lists one episode involving an unspecified arsenical in a fish kill in 1973. An unknown number of fish

in a pond near a soybean field were killed. The pond had apparently been contaminated by waste cans and containers of pesticides, including linuron, alachlor, trifluralin, and paraquat. The arsenic content of the water was 73 ppm. There was insufficient evidence either to prove or disprove involvement of arsenic.

Table 27 shows the acute toxicities of sodium arsenite and arsenate to various aquatic species. Other studies indicate possible adverse effects of inorganic arsenic to aquatic organisms at levels below acute toxicity concentrations.

Table 27. Acute Toxicity of Sodium Arsenite and Arsenate to Fresh Water and Marine/Estuarine Species (in ppm)

Sodium Arsenite	24-hr	48-hr	96-hr	Reference
Daphnia sp.		9.1		Mackenthun 1960
Water flea (*Daphnia pulex*)		1.8		Cope 1966
Stonefly nymph (*Pteronarcys californicus*)		80		Cope 1966
Water flea (*Simocephalus serrulatus*)		1.4		Cope 1966
Channel catfish fingerlings	47.9	25.9	25.9	Clemens & Sneed 1959
Channel catfish-juvenile (*Ictalurus punctatus*)		56.9	31.2	Cardwell et al. 1976
Rainbow trout		60		Crosby & Tucker 1966
Rainbow trout (*Salmo gairdneri*)		36.5		Cope 1966
Bluegill (*Lepomis macrochirus*)		44		Cope 1966
Bluegill		44		Crosby & Tucker 1966
Bluegill-juvenile (*Lepomis macrochirus*)			72	Cardwell et al. 1976
Brook trout (*Salvelinus fontinalis*)	53.9	27.8	25.8 a/	Cardwell et al. 1976
Fathead minnows-juvenile (*Pimephales promelas*)	36.2	31.3	27	Cardwell et al. 1976
Goldfish-juvenile (*Carassius auratus*)	89.6	54.6	44.9	Cardwell et al. 1976
Flagfish fry (*Jordanella floridae*)	69.8 b/		48.5 a/	Cardwell et al. 1976
Minnow (*Notropsis hudsonius*)	45	29	72 c/	Boschetti & McLoughlin 1957
Tadpole (*Adelotus brevis*)	152	119	96	Johnson 1976
Tadpole (*Limnodynastes peroni*)	108	92	60	Johnson 1976
Tadpole (*Bufo marinus*)	195	150	123	Johnson 1976
Juvenile bay scallops (*Argopecten irradians*)				
Oyster embryos (*Crassostrea virginica*)	6.50	4.40	3.49	Nelson et al. 1976
		7.50		Calabrese et al. 1973
Sodium Arsenate				
Green sunfish (*Lepomis cyanellus*)	175	150		Sorensen 1976a
Daphnia sp.		20		Mackenthun 1960
Goldfish (*Carassius auratus*)		32		Weir & Hine 1970

a/ 93-hr LC-50.
b 29-hr LC-50.
c/ 72-hr TL-50.

A study by Wiebe (1930) concluded that fingerling stages of four species of game fishes and three species of forage fishes exposed to concentrations of sodium arsenite ranging from 2 to 7 ppm for varying periods of time did not show any harmful effects. Species used in the study were largemouth black bass (*Micropterus salmoides*), smallmouth black bass (*M. dolomieu*), white crappie (*Pomoxis annularis*), bluegill (*Lepomis incisor*), golden shiner (*Notemigonus crysoleucas*), goldfish (*Carassius auratus*), and bullhead (*Ameiurus nebulosus*).

Since copper acetoarsenite is insoluble in water, it is generally regarded as nontoxic to fish. Bishop (1940) reported on the effects of copper acetoarsenite for mosquito control in Wheeler Reservoir in Tennessee, applied at about one pound per acre. The author noted that treatments showed no deleterious effect upon vegetation; there was no evidence of a "catastrophic destruction" of aquatic organisms important as fish food; and soil samples showed accumulation of arsenical residues in variable amounts in the bottom of reservoirs.

In another study on copper acetoarsenite, McCormick (1940) attempted to determine the amount of arsenic exposure to fish from mosquito-control dusting. Fish used in

the study were mosquitofish (Gambusia patruelis), catfish (Ameiurus melas), killifish (Fundulus dispar), and silversides (Labidesthes sicculus). Fish were treated with 2 to 6 grams of pure or mixture paris green. Out of 15 series of tests, two were discarded because of procedural errors. Of the remaining 13 series of tests, eight were listed as showing positive results for arsenic residues, and five were negative. Of these, however, two series which showed positive results were rejected because the fish had not been adequately washed, and particles of copper acetoarsenite in the water could have adhered to their skin; one negative result was also rejected because tests were run only on very small, individual fish. Amounts of arsenic found ranged from trace to 0.1 mg. The author noted that Gambusia were able to withstand total dosages of 18 and 20 grams of copper aceto-arsenite (56 to 60 ppm arsenic trioxide).

Grindley (1946) studied the effects of sodium arsenite and sodium arsenate on minnows (Phoxinus phoxinus). Toxicity to fish was determined by when they had permanently overturned (i.e., lost equilibrium). At a concentration 47.5 ppm sodium arsenite, it took an average of 19 hours before fish had overturned; at 17.8 ppm, it took an average of 36 hours.

At 234 ppm sodium arsenate, it took an average of 15.85 hours before fish had overturned; at 610 ppm, it took an average of 9.05 hours. None of the fish exposed to sodium arsenite or sodium arsenate recovered after removal to fresh water. The author also noted that "smaller concentrations [of arsenic] acting over long periods in a river would [probably] damage a fishery, either by their continued action on the fish themselves or by their toxic action on other aquatic organisms which serve as the food of the fish."

Gilderhus (1966) studied some effects of sublethal concentrations of sodium arsenite on adult and immature bluegills (Lepomis macrochirus). Chronic toxicity to fish was based on survival, growth, hematocrit measurements, ratio of gonad weight to body weight, and pathological changes in tissues. Nine concrete pools were treated with varying concentrations of sodium arsenite over a 16-week period.

Neither hematocrit measurements nor gonad-to-body weight ratios could be related to arsenic concentration.

Survival, however, was definitely affected. In immature bluegills, survival decreased as the amount of arsenic treatment increased. Only 18% of immature fish

survived after 16 weeks in a pool receiving a total treatment of 19.2 ppm sodium arsenite, compared to 90% survival in the control pool. Survival of immature fish in pools receiving totals of 1.2, 4.0, and 4.8 ppm sodium arsenite was also reduced compared to controls (54, 52, and 45%, respectively). Survival of adult bluegill was 31% in the pool receiving 19.2 ppm, compared to 60% in controls. Survival of adults in all other treated pools ranged from 50 to 62%.

Growth (length and weight gain) for immature bluegill declined as sodium arsenite concentrations increased. Adult bluegills remained about the same length during the experiment, but adults at the four highest concentrations gained less weight, on average, by four to six grams (25 to 37% of their weight at the experiment's start). Control fish, however, did not have a higher final weight than fish from some treated pools. The author could not account for this but speculated that light arsenic treatment enhanced conditions for growth.

Only adult fish from the control pool and fish from pools which received weekly treatments were examined histopathologically. Exposed fish had more hemorrhagic globes on the gills than controls. Among other symptoms noted in some exposed fish, and not seen in control fish, were fat infiltration of the liver, vacuolation of the

cytoplasm of hepatic cells, foci of necrosis, and a massive extravasation of erythrocytes, which lifted the lining of the ventricle of the heart, along with interventricular fibrin and necrotic cells. In addition, in ovaries of fish from heavily treated pools, 20 to 50% of the oogonia and primary and secondary oocytes appeared as encapsulated cells. Mature ova appeared normal, but ovaries contained fewer packed cells than normal organs.

The author noted that applications of 4.0 ppm or more of sodium arsenite in one year inhibited growth of both adult and immature bluegills, and adversely affected survival of immatures. Among adults, residues of about 5.0 ppm in flesh were associated with severe loss of weight and high mortality; growth was slowed slightly with residues in flesh between 1 and 3 ppm.

Weir and Hine (1970) studied impairment of conditioned behavior of goldfish (Carassius auratus) by various metals, including sodium arsenate. Goldfish conditioned to avoid electric shocks were exposed to sublethal amounts of sodium arsenate for 24 and 48 hours. The authors found that the lowest arsenic concentration which significantly impaired conditioned behavior was 0.1 ppm, or 1/320 of the LC-50 and 1/15 of the LC-1. The authors noted that deleterious

effects [i.e., impairment of conditioned behavior] were demonstrated below the arsenic levels currently accepted as safe.

Sorensen (1976b) studied thermal effects on accumulation of arsenic in green sunfish (Lepomis cyanellus). Nine groups of 20 fish each were acclimated in aquaria at 10°C [=50°F], 20°C [-68°F], and 30°C [=86°F] (three groups for each temperature level). One group at each temperature was then exposed to 0, 30, and 60 ppm sodium arsenate for five weeks. Every third day, the fish were placed in fresh test solutions. At weekly intervals for five weeks, two live fish were collected at random from each of the nine treatments. Liver, right muscle fillet, and guts (without contents) were analyzed for arsenic content. (For results of these analyses, see Table 28.)

Sorensen reported that the LT-50 (times for 50% mortality), at the same arsenic concentration, increased as temperature increased. At 60 ppm arsenic, the LT-50 at 10°C was 678 hours, at 20°C 210 hours, and at 30°C 124 hours. At 30 ppm arsenic, the LT-50 values at 20 and 30°C were 527 and 209 hours, respectively. The author concluded that arsenic uptake in liver, gut, and muscle increased with increasing temperature, arsenic concentration, and length of exposure.

Table 28 . Tissue uptake of arsenic (ug/mg wet weight)[a/] by the green sunfish

Degree C and arsenic concentration in ppm	Liver Uptake Weeks 1st	2nd	3rd	4th	5th
10°C; 0 ppm	0.03	0.03	0.03	0.03	0.03
10°C; 30 ppm	0.03	0.06	0.12	0.12	0.21
10°C; 60 ppm	0.03	0.09	0.135	0.15	0.24
20°C; 0 ppm	0.03	0.03	0.03	0.03	0.03
20°C; 30 ppm	0.15	0.225	0.574	1.033	0.743
20°C; 60 ppm	<0.364	0.424	----[b/]	----[b/]	----[b/]
30°C; 0 ppm	0.03	0.03	0.03	0.03	0.03
30°C; 30 ppm	<0.364	0.334	----[b/]	----[b/]	----[b/]
30°C; 60 ppm	0.545	1.393	----[b/]	----[b/]	----[b/]
Gut Uptake					
10°C; 0 ppm	ND	ND	0.01	0.02	0.01
10°C; 30 ppm	0.03	0.05	0.1	0.141	0.159
10°C; 60 ppm	0.06	0.05	0.133	0.126	0.159
20°C; 0 ppm	ND	ND	0.005	0.005	0.01
20°C; 30 ppm	0.01	0.61	0.221	0.247	0.221
20°C; 60 ppm	0.65	0.226	----[b/]	----[b/]	----[b/]
30°C; 0 ppm	ND	ND	ND	ND	ND
30°C; 30 ppm	<0.126	0.035	----[b/]	----[b/]	----[b/]
30°C; 60 ppm	0.75	0.242	----[b/]	----[b/]	----[b/]
Muscle Uptake					
10°C; 30 ppm	ND	NM	NM	ND	0.029
10°C; 60 ppm	0.028	0.047	0.013	NM.	0.41
20°C; 30 ppm	0.023	0.02	0.025	0.037	0.039
20°C; 60 ppm	0.017	0.015	0.015	----[b/]	----[b/]
30°C; 30 ppm	0.02	0.053	----[b/]	----[b/]	----[b/]
30°C; 60 ppm	0.032	0.04	----[b/]	----[b/]	----[b/]

a/ Values derived from author's graphs and may not be exact.
b/ No fish remained alive at these concentrations for these time periods.
ND = Not detected; NM = Not measured.

The Agency points out that, particularly for aquatic food organisms (phytoplankton, zooplankton, and invertebrates), arsenic produced the adverse effects listed by Menhinick (1962), even at sublethal concentrations. The Agency also notes the possibility of long-term adverse effects on larger aquatic organisms from sublethal arsenic concentrations. The Agency is concerned that indirect contaminations of water from use of arsenical pesticides could have long-term adverse effects on aquatic organisms through reduction in numbers of food organism and oxygen-producing algae (Lawrence 1958; Cowell 1965; Ball and Hooper 1966; and Gilderhus 1966) and through adverse effects on survival ability of larger aquatic organisms (Gilderhus 1966; Weir and Hine 1970).

(2) Population Reductions in Nontarget Mammalian Wildlife

Swiggart et al. (1972) reported on eleven dead deer (8 females, 3 males) that were found in a 600 acre area of Tennessee, which recently had been treated with an arsenical herbicide containing 39.6% arsenic acid. The product was applied aerially to agricultural land for control of Johnson grass at the rate of five gallons of mixture per acre (prepared in the ratio of 1 gallon of herbicide to 9 gallons of water). This particular use of the pesticide violated the USDA registration of the product.

Qualitative and quantitative analyses by laboratories showed a high level of arsenic in deer tissue specimens and in samples of water, soil, and vegetation from the area.

Boyce and Verme (1954) studied the effect of sodium arsenite, used as a tree debarking agent, on wildlife mortality in both commercially treated plots and controlled study areas. In several debarking test plots set up by lumber companies in Michigan, a number of deer, porcupines, and snowshoe hares were found dead. Analyses of the digestive organs indicated arsenic as the cause of death. Several controlled experiments were done on confined deer exposed to trees girdled and treated with sodium arsenite. In some studies, deer remained healthy and apparently did not lick the treated trees. In similar studies, some deer died from arsenic poisoning as shown by tissue analyses.

In a mineral preference test, two groups of adult and yearling does were exposed to trees treated with sodium, potassium, and ammonium arsenite, as well as sodium chloride. Sodium arsenite was only slightly more preferred than the other compounds, including sodium chloride. In another study, potassium arsenite was used instead of the sodium salt to determine if one form is more attractive to deer. No deer were found dead. Penned deer were exposed to sugar cubes dyed various colors to determine if the red dye added to commercial sodium arsenite

formulations was attractive to deer. Deer showed only a slight preference for the red cubes.

From toxicity tests on confined deer, it was determined that only small amounts of arsenite were required to produce mortality. Death usually occurred within a relatively short time after ingestion. In one test, a yearling doe died 12 hours after ingesting a total of 4 cc of arsenite (potassium and sodium salts) in food pellets. Ingestion of foliage from treated trees caused no observable ill effects.

The animals apparently did not obtain the sodium arsenite from the girdled area of the trees but came in contact with the spillage at the base of the trees.

References*

Albert, W.B., and C.H. Arndt. 1931. The concentration of soluble arsenic as an index of arsenic toxicity to plants. S.C. Agric. Exp. Stat. Ann. Rep. 44:47-48.

Albert, R.E., R.E. Train, and E. Anderson. 1977. Rationale developed by the Environmental Protection Agency for the assessment of carcinogenic risks. J. Natl. Cancer Inst. 58(5):1537-1541.

Anastasia, F.B., and W.J. Kender. 1973. The influence of soil arsenic on the growth of lowbush blueberry. J. Environ. Qual. 2(3):335-337.

Anke, M., M. Grun, and M. Partschefeld. 1976. The essentiality of arsenic for animals. Pages 403-409 in D.D. Hemphill, ed., Trace substances in environmental health, Vol. X. Univ. of Missouri, Columbia, Missouri.

Arsenault, R.D. 1975. Public hearings: proposed exposure standard for inorganic arsenic [April 15, 1975, Washington, D.C.]. Pages 1015-1260 in stenographic transcript of hearings. Federal Data Retrieval Systems, Inc., Arlington, Va.

Arsenault, R.D. 1977. Health aspects of C.C.A. wood preservatives - a review of arsenates and cancer. Koppers Company, Inc., Pittsburgh, Pa.

Ashton, F.M., and A.S. Crafts. 1973. Mode of action of herbicides. John Wiley and Sons, Inc., New York. 504 pp.

AWPI. American Wood Preservers Institute. 1976. All weather wood foundation system: information and response to concrete industry allegations, Chapter E. (Unpublished.)

AWPI. American Wood Preservers Institute. 1977. Arsenical wood preservatives. American Wood Preservers Institute, McLean, Virginia.

Ball, R.C., and F.H. Hooper. 1966. Use of ^{74}As-tagged sodium arsenite in a study of effects of a herbicide on pond ecology. Pages 149-163 in Isotopes in weed research. International Atomic Energy Agency, Vienna.

* Copies of specific references will be provided on request. There will be a charge to cover duplication costs for such requests. A copy of the Position Document and all references are available for public inspection in the Office of Special Pesticide Reviews (WH-566), Office of Pesticide Programs, Environmental Protection Agency, East Tower, Room 447, 401 M Street S.W., Washington, D.C. 20460.

Baron, D., I. Kunick, I. Frischmuth, and J. Petres. 1975. Further in vitro studies on the biochemistry of the inhibition of nucleic acid and protein synthesis induced by arsenic. Arch. Derm. Res. 253:15-22.

Beaudoin, A.R. 1974. Teratogenicity of sodium arsenate in rats. Teratology 10:153-158.

Bennett, R.L., and M.H. Malamy. 1970. Arsenate resistant mutants of Escherichia coli and phosphate transport. Biochem. Biophys. Res. Commun. 40(2):496-503.

Bergoglio, R.M. 1964. Mortality from cancer in regions of arsenical waters of the province of Cordoba Argentine Republic. (Transl. from Spanish.) Pren. Med. Argent. 51:994-998.

Bishop, E.L. 1940. Cooperative investigations of the relation between mosquito control and wildlife conservation. Science 92(2383):201-202.

Bishop, R.F., and D. Chisholm. 1962. Arsenic accumulation in Annapolis Valley orchard soils. Can. J. Soil Sci. 42:77-80.

Blus, L.J., B.S. Neely, Jr., T.G. Lamont, and B. Mulhern. 1977. Residues of organochlorines and heavy metals in tissues and eggs of brown pelicans, 1969-73. Pest. Mon. J. 11(1):40-53.

Boschetti, M.M., and T.F. McLoughlin. 1957. Toxicity of sodium arsenite to minnows. Sanitalk 5(4):14-18.

Bowen, H.J.M. 1966. Trace elements in biochemistry. Academic Press, London.

Boyce, A.P., and L.J. Verme. 1954. Toxicity of arsenite debarkers to deer in Michigan. Report No. 2025, presented at the 16th Midwest Wildlife Conference, St Louis, Missouri. (Unpublished.)

Bradicich, R., N.E. Foster, F.E. Hons, M.T. Jeffus, and C.T. Kenner. 1969. Arsenic in cottonseed products and various commodities. Pest. Mon. J. 3(3):139-141.

Braman, R.S. 1975. Arsenic in the environment. Pages 108-123 in E.A. Woolson, ed., Arsenical pesticides. ACS Symposium Series 7. American Chemical Society, Washington, D.C.

Braun, W. 1958. Carcinoma of the skin and the internal organs caused by arsenic. Ger. Med. Monthly 3(10):321-324.

Budy, A.M., and M.N. Rashad. 1976. Cancer mortality among carpenters in Hawaii. Presented at 3rd Intntl. Symp. on Detection and Prevention of Cancer, New York. (Unpublished.)

Burgdorf, W., K. Kurvink, and J. Cervenka. 1977. Elevated sister chromatid exchange rate in lymphocytes of subjects treated with arsenic. Hum. Genet. 36:69-72.

Burk, D., and A.R. Beaudoin. 1977. Arsenate-induced renal agenesis in rats. Teratol. 16(3):247-260.

Button, D.K., and S.S. Dunker. 1971. Biological effects of copper and arsenic pollution. U.S. Environmental Protection Agency, Washington, D.C. Report Number R71-8, 59pp.

Byron, W.R., G.W. Bierbower, J.B. Brouwer, and W.H. Hansen. 1967. Pathologic changes in rats and dogs from two-year feeding of sodium arsenite or sodium arsenate. Toxicol. Appl. Pharmacol. 10:132-147.

Calabrese, A., R.S. Collier, D.A. Nelson, and J.R. MacInnes. 1973. The toxicity of heavy metals to embryos of the American oyster Crassostrea virginica. Mar. Biol. 18:162-166.

Cardwell, R.D., D.G. Foreman, T.R. Payne, and D.J. Wilbur. 1976. Acute toxicity of selected toxicants to six species of fish. U.S. Environmental Protection Agency, Environmental Research Laboratory, Duluth, Minnesota.

Carey, A.E., G.B. Wiersma, and H. Tai. 1976. Pesticide residues in urban soils from 14 United States cities, 1970. Pest. Mon. J. 10(2):54-60.

Case, A.A. 1974. Arsenics. Pages 217-231 in Environmental chemicals, human and animal health, 3rd Annual Conference Proceedings, Colorado State University, Ft. Collins, Colorado. U.S. Environmental Protection Agency, Washington, D.C.

Casto, B.C., W.J. Pieczynski, R.L. Nelson, and J.A. DiPaolo. 1976. In vitro transformation and enhancement of viral transformation with metals. (Abstract). Proc. Amer. Assoc. of Cancer Res. 17:12.

Casto, B.C., J. Meyers, and J.A. DiPaolo. 1977. Assay of industrial chemicals in Syrian hamster cells for enhancement of viral transformation. (Abstract). Proc. Amer. Assoc. of Cancer Res. 18:155.

Chapman, A.C. 1926. On the presence of compounds of arsenic in marine crustaceans and shell fish. Analyst 51:548-563.

Clemens, H.P., and K.E. Sneed. 1959. Lethal doses of several chemicals for fingerling channel catfish: special scientific report - fisheries no. 316. U.S. Dept. of the Interior, Washington, D.C.

Cooper, H.P., W.R. Paden, E.E. Hall, W.B. Albert, W.B. Rogers, and J.A. Riley. 1931. Effect of calcium arsenate on the productivity of certain soil types. S.C. Agric. Exp. Stat. Ann. Rep. 44:28-36.

Cooper, H.P., W.R. Paden, E.E. Hall, W.B. Albert, W.B. Rogers, and J.A. Riley. 1932. Soils differ markedly in their response to additions of calcium arsenate. S.C. Agric. Exp. Stat. Ann. Rep. 45:23-27.

Cope, O.B. 1966. Contamination of the freshwater ecosystem by pesticides. J. Appl. Ecol. 3(Suppl.):33-44.

Coulson, E.J., R.E. Remington, and K.M. Lynch. 1935. Metabolism in the rat of the naturally occurring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10(3): 255-270.

Cowell, B.C. 1965. The effects of sodium arsenite and silvex on the plankton populations in farm ponds. Trans. Am. Fish. Soc. 94(4):371-377.

Cox, D.P. 1975. Microbiological methylation of arsenic. Pages 81-96 in E.A. Woolson, ed., Arsenical pesticides. ACS Symposium Series 7. American Chemical Society, Washington, D.C.

Crawford, T.B.B., and I.D.E. Storey. 1944. A quantitative micro-method for the separation of inorganic arsenite from arsenate in blood and urine. Biochem. J. 38:195-198.

Crecelius, E.A. 1977a. Arsenite and arsenate levels in wine. Bull. Environ. Contam. Tox. 18(2):227-230.

Crecelius, E.A. 1977b. Changes in the chemical speciation of arsenic following ingestion by man. Environ. Health Persp. 19:147-150.

Crecelius, E.A., C.J. Johnson, and G.C. Hofer. 1974. Contamination of soils near a copper smelter by arsenic, antimony and lead. Water, Air, Soil Pollut. 3:337-342.

Crosby, D.G., and R.K. Tucker. 1966. Toxicity of aquatic herbicides to Daphnia magna. Science 154(3746):289-291.

Cummings, J.G. 1966. Pesticides in the total diet. Pages 30-45 in F.A. Gunther, ed., Residue reviews, Vol. 16. Springer-Verlag, New York.

Denk, R., H. Holzmann, H.J. Lange, and D. Greve. 1969. Evidence of delayed arsenic injuries in autopsied Mosel winegrowers. (Transl. from German.) Med. Welt. 20(11): 557-567.

Deuel, L.E., and A.R. Swoboda. 1972. Arsenic toxicity to cotton and soybeans. J. Environ. Qual. 1(3):317-320.

Dreher, W.A. 1974. Report on hand wipe tests for arsenic on CCA treated plywood, activity 1646. Koppers Co., Inc., Monroeville, Pa. (Unpublished.)

Ducoff, H.S., W.B. Neal, R.L. Straube, L.O. Jacobson, and A.M. Brues. 1948. Biological studies with arsenic[76]. II. excretion and tissue localization. Proc. Soc. Exp. Biol. (N.Y.) 69:548-554.

Duggan, R.E., and J.R. Weatherwax. 1967. Dietary intake of pesticide chemicals. Science 157:1006-1010.

Duggan, R.E., H.C. Barry, and L.Y. Johnson. 1966. Pesticide residues in total-diet samples. Science 151:101-104.

Duggan, R.E., H.C. Barry, and L.Y. Johnson. 1967. Pesticide residues in total diet samples (II). Pest. Mon. J. 1(2):2-12.

Duncan, L.J., E.L. Keitz, E.P. Krajeski. 1973. Selected characteristics of hazardous pollutant emission. Vol. II. Mitre Corporation Publication No. MTR-6401, 331 pp.

EPA. U.S. Environmental Protection Agency. 1971a. PR Notice 71-7. Loose-leaf pub. 1 p.

EPA. U.S. Environmental Protection Agency. 1971b. PR Notice 71-3. Loose-leaf pub. 8 pp.

EPA. U.S. Environmental Protection Agency. 1972. PR Notice 72-4. Loose-leaf pub. 3 pp.

EPA. U.S. Environmental Protection Agency, Special Pesticides Review Groups. 1975. Arsenical pesticides, man, and the environment. Washington, D.C.

EPA. U.S. Environmental Protection Agency, Office of Air and Waste Management. 1976a. Air pollutant assessment report on arsenic. Research Triangle Park, North Carolina.

EPA. U.S. Environmental Protection Agency, Office of Toxic Substances. 1976b. Technical and microeconomic analysis of arsenic and its compounds. Washington, D.C.

EPA. U.S. Environmental Protection Agency, Office of Pesticide Programs, Criteria and Evaluation Division. 1976c. Arsenical pesticides: internal review. 210p. (Unpublished).

EPA. U.S. Environmental Protection Agency, Pesticide Episode Response Branch. 1977. Episode summary for reports involving arsenicals: pesticide episode review system report no. 94. Washington, D.C. (unpublished).

EPA. U.S. Environmental Protection Agency, Carcinogen Assessment Group. 1978. The Carcinogen Assessment Group's preliminary report on inorganic arsenic. (Unpublished.)

Epps, E.A., and M.B. Sturgis. 1939. Arsenic compounds toxic to rice. Soil Sci. Soc. Amer. Proc. 4:215-218.

Fahlstrom, G.B., P.E. Gunning, and J.A. Carlson. 1967. Copper-chrome-arsenate wood preservatives: a study of the influence of composition on leachability. Forest Prod. J. 17(7):17-22.

Fairhall, L.T., and P.A. Neal. 1938. The absorption and excretion of lead arsenate in man. Pub. Health Rpts. 53(29):1231-1245.

Ferguson, J.F., and J. Gavis. 1972. A review of the arsenic cycle in natural waters. Water Research 6:1259-1274.

Ferm, V.H., and S.J. Carpenter. 1968. Malformations induced by sodium arsenate. J. Reprod. Fert. 17:199-201.

Ferm, V.H., A. Saxon, and B.M. Smith. 1971. The teratogenic profile of sodium arsenate in the golden hamster. Arch. Environ. Health. 22:557-560.

Fiscor, G., and G.M. nii Lo Piccolo. 1972. Survey of pesticides for mutagenicity by the bacterial-plate assay method. EMS Newsl. 6:6-8.

Franke, K.W., and A.L. Moxon. 1936. A comparison of the minimum fatal doses of selenium, tellurium, arsenic and vanadium. J. Pharmacol. Exp. Therap. 58:454-459.

Franke, K.W., A.L. Moxon, W.E. Poley, and W.C. Tully. 1936. Monstrosities produced by the injection of selenium salts into hens' eggs. Anatom. Rec. 65:15-22.

Frohn, W. 1938. Social medicine and hygiene: occupational arsenic poisoning of wine growers. (Transl. from German.) Munchener Med. Wochenschrift 42:1630-1635.

Frost, D.V. 1967. Arsenicals in biology -- retrospect and prospect. Fed. Proc. Fed. Amer. Soc. Exp. Biol. 26:194-208.

Galy, P., R. Touraine, J. Brune, R. Roudier, and P. Gallois. 1963. Pulmonary cancer of arsenical origin in the winegrowers of the Beaujolais. (Transl. from French.) Fr. J. Thoracic Med. Surg. 17:303-311.

Geisman, J.R., W.E. Carey, W.A. Gould, and E.K. Alban. 1969. Distribution of arsenic residues by activation analysis. J. Food Sci. 34:295-298.

Gilderhus, P.A. 1966. Some effects of sublethal concentrations of sodium arsenite on bluegills and the aquatic environment. Trans. Am. Fish. Soc. 95(3): 289-296.

Ginsburg, J.M. 1965. Renal mechanism for excretion and transformation of arsenic in the dog. Am. J. Physiol. 208(5):832-840.

Ginsburg, J.M., and W.D. Lotspeich. 1963. Interrelations of arsenate and phosphate transport in the dog kidney. Am. J. Physiol. 205(4):707-714.

Goudey, R.F. 1946. Chemical weed control. J. Am. Water Works Assoc. 38:186-202.

Greaves, J.E. 1934. The arsenic content of soils. Soil Sci. 38(5):355-362.

Grindley, J. 1946. Toxicity to rainbow trout and minnows of some substances known to be present in waste water discharged to rivers. Ann. Appl. Biol. 33:103-112.

Harvey, S.C. 1975. Heavy metals. Pages 924-928 in L.S. Goodman and A. Gilman, eds., The pharmacological basis of therapeutics, 5th ed. Macmillan Pub. Co., Inc., New York.

HEW. U.S. Department of Health, Education, and Welfare. 1967. Control and disposal of cotton-ginning wastes. A Symposium, Dallas, Texas, May 3-4, 1966. Public Health Service Publication 999-Ap-31. Cincinnati, Ohio.

HEW. U.S. Department of Health, Education, and Welfare. 1969. Report of the Secretary's commission on pesticides and their relationship to environmental health. Washington, D.C.

Hill, A.B., and E.L. Faning. 1948. Studies in the incidence of cancer in a factory handling inorganic compounds of arsenic: I. mortality experience in the factory. Brit. J. Industr. Med. 5(1):1-6.

Holmberg, R.E., Jr., and V.H. Ferm. 1969. Interrelationships of selenium, cadmium, and arsenic in mammalian teratogenesis. Arch. Environ. Health 18(6):873-877.

Hood, R.D. 1972. Effects of sodium arsenite on fetal development. Bull. Environ. Contam. Toxicol. 7(4): 216-222.

Hood, R.D., and S.L. Bishop. 1972. Teratogenic effects of sodium arsenate in mice. Arch. Environ. Health 24:62-65.

Hood, R.D., and C.T. Pike. 1972. BAL alleviation of arsenate-induced teratogenesis in mice. Teratology 6:235-238.

Hood, R.D., G.T. Thacker, and B.L. Patterson. Undated. Prenatal effects of oral versus intraperitoneal sodium arsenate in mice. (In Press, J. Environ. Path. and Toxicol.)

Hood, R.D., G.T. Thacker, and B.L. Patterson. 1977. Effects in the mouse and rat of prenatal exposure to arsenic. Environ. Health Perspect. 19:219-222.

Hunter, F.T., A.F. Kip, and J.W. Irvine, Jr. 1942. Radioactive tracer studies on arsenic injected as potassium arsenite. I. excretion and localization in tissues. J. Pharmacol. Exp. Therap. 76:207-220.

Irgolic, K.J. 1978. Analysis of water samples from Antofagasta, Chile; Pei Men and Pu Tai, Taiwan; Bakersfield, California and Nova Scotia, Canada. Quarterly Progress Report, "Speciation of Arsenic in Water Supplies." U.S. Environmental Protection Agency, Washington, D.C. (Unpublished.)

Iwataki, N., and K. Horiuchi. 1959. Concering [sic] the upper limits of arsenic in blood, urine and feces among normal Japanese population. Osaka City Med. J. 5(2):209-217.

Jacobs, L.W., and D.R. Keeney. 1970. Arsenic - phosphorous interactions on corn. Commun. Soil Sci. Plant Anal. 1(2):85-93.

Jacobs, L.W., D.R. Keeney, and L.M. Walsh. 1970. Arsenic residue toxicity to vegetable crops grown on Plainfield sand. Agron. J. 62:588-591.

James, L.F., V.A. Lazar, and W. Binns. 1966. Effects of sublethal doses of certain minerals on pregnant ewes and fetal development. Amer. J. Vet. Res. 27(116):132-135.

Johanson, R., and F.A. Dale. 1973. Arsenic on the surface of round pine treated with Cu-Cr-As preservative. Holzforschung 27:187-189.

Johnson, C.R. 1976. Herbicide toxicities in some Australian anurans and the effect of subacute dosages on temperature tolerance. Zoolog. J. Linn. Soc. 59:79-83.

Jones, J.S., and M.B. Hatch. 1945. Spray residues and crop assimilation of arsenic and lead. Soil Sci. 60:277-288.

Jung, E.G., and B. Trachsel. 1970. Molecular-biological studies of arsenic carcinogenesis. Arch. Klin. Exp. Derm. 237:819-826.

Jung, E.G., B. Trachsel, and H. Immich. 1969. Arsenic as an inhibitor of the enzymes concerned in cellular recovery (dark repair). Germ. Med. Monthly 14:614-616.

Kerr, T.W., Jr. 1941. Sand-arsenical mixtures for controlling white grubs and their effect upon the growth of strawberries. J. Econ. Entomol. 34(3):405-411.

Klemmer, H.W., E. Leitis, and K. Pfenninger. 1975. Arsenic content of house dusts in Hawaii. Bull. Environ. Contam. Toxicol. 14(4):449-452.

Kojima, H. 1974. Developmental pharmacology of arsenite. II. effect of arsenite on pregnancy, nutrition, and hard tissue. (Transl. from Japanese.) Nippon Yakurigaku Zasshi 70(2):149-163.

Kuratsune, M., S. Tokudome, T. Shirakusa, M. Yoshida, Y. Tokumitsu, T. Hayano, and M. Seita. 1974. Occuaptional lung cancer among copper smelters. Intntl. J. Cancer 13:552-558.

Lakso, J.U., and S.A. Peoples. 1975. Methylation of inorganic arsenic by mammals. J. Agr. Food Chem. 23(4):674-676.

Landau, E., D.J. Thompson, R.G. Feldman, G. Goble, and W.J. Dixon. 1977. Selected non-carcinogenic effects of industrial exposure to inorganic arsenic. U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C.

Lanz, H., Jr., P.C. Wallace, and J.G. Hamilton. 1950. The metabolism of arsenic in laboratory animals using As^{74} as a tracer. Univ. Calif. Pub. in Pharm. 2(20):263-282.

Lawrence, J.M. 1958. Recent investigations of the use of sodium arsenite as an algacide and its effects on fish production in ponds. Proc. 11th Ann. Conf. S.E. Assoc. Game and Fish Comm., pp. 281-287.

Lee, A.M., and J.F. Fraumeni, Jr. 1969. Arsenic and respiratory cancer in man: an occupational study. J. Natl. Cancer Inst. 42(6):1045-1052.

Lee, S.S., B.R. Holt, and E.A. Woolson. 1977. Environmental mass balance of arsenic. IV. simulations of arsenic transformations in water and soil. U.S. Environmental Protection Agency. SRI/CRESS Rep. No. 4.

Lehman, A.J. 1962. The annual per capita consumption of selected items of food in the United States. Qtly. Bull. Assn. Food & Drug Office 26(3):149-151.

Letter. 1975. [No title], dated April 14, 1975. From Warren S. Ferguson, Director, Occupational Health and Product Safety, to Mr. R.D. Arsenault, Technical Director, Koppers Company, Inc.

Letter. 1977a. [No title], dated May 31, 1977. From Bruce C. Casto, Sc.D., Biolabs, Inc., to Dr. Bill Waugh, [U.S. Environmental Protection Agency].

Letter. 1977b. [No title], dated December 5, 1977. From Carole A. Kimmel, Natl. Center for Toxicological Research, HEW, to Anita L. Schmidt, Project Manager, Office of Special Pesticide Reviews.

Letter. 1978. [No title], dated March 6, 1978. From Wataru Takahashi, University of Hawaii at Manoa, to Mr. W.T. Waugh, Project Manager, Office of Special Pesticide Reviews.

Lincer, J.L. 1975. The impact of synthetic organic compounds on estuarine organisms. Pages 425-435 in U.S. Environmental Protection Agency, Estuarine pollution control and assessment: proceedings of a conference, Volume II. Washington, D.C.

Lombardini, J.B. 1978a. Validation report on Crawford and Storey (1944) [q.v.]. (Unpublished.)

Lombardini, J.B. 1978b. Validation report on Ginsburg and Lotspeich (1963) [q.v.]. (Unpublished.)

Lombardini, J.B. 1978c. Validation report on Ginsburg (1965) [q.v.]. (Unpublished.)

Lu, F-J., C-Kk. Yang, and K-H. Ling. 1975. Physico-chemical characteristics of drinking water in blackfoot endemic areas in Chia-I and Tainan Hsiens. J. Formosan Med. Assn. 74(9):596-605.

Lu, F-J., M-H. Tsai, and K-H. Ling. 1977a. Studies on fluorescent compound in drinking water of blackfoot disease endemic areas: 1. the toxic effects of fluorescent compound on the chick embryos. (Transl. from Chinese.) J. Formosan Med. Assn. 76(1):58-63.

Lu, F-J., M-H. Tsai, and K-H. Ling. 1977b. Studies on fluorescent compounds in drinking water of blackfoot endemic areas: 2. isolation and identification of fluorescent compounds. (Transl. from Chinese.) J. Formosan Med. Assn. 76(3):209-217.

Lucas, H.F., Jr., D.N. Edgington, and P.J. Colby. 1970. Concentration of trace element in Great Lakes fishes. J. Fish. Res. Bd. Canada 27:677-684.

Lunde, G. 1969. Water soluble arseno-organic compounds in marine fishes. Nature 224:186-187.

Lunde, G. 1970. Analysis of arsenic and selenium in marine raw materials. J. Sci. Food Agric. 21:242-247.

Lunde, G. 1972. The analysis of arsenic in the lipid phase from marine and limnetic algae. Acta Chem. Scand. 26:2642-2644.

Lunde, G. 1973a. Separation and analysis of organic-bound and inorganic arsenic in marine organisms. J. Sci. Food Agric. 24:1021-1027.

Lunde, G. 1973b. The synthesis of fat and water soluble arseno organic compounds in marine and limnetic algae. Acta Chem. Scand. 27:1586-1594.

Lynch, D.W. 1973. Selected toxic metals in Ohio's upland wildlife. Master's Thesis, Ohio State University.

Machlis, L. 1941. Accumulation of arsenic in the shoots of Sudan grass and bush bean. Plant Physiol. 16:521-544.

Mackenthun, K.M. 1960. Some limnological investigations on the long-term use of sodium arsenite as an aquatic herbicide. Proc. 17th N. Cent. Weed Control Conf.

MacPhee, A.W., D. Chisholm, and C.R. MacEachern. 1960. The persistence of certain pesticides in the soil and their effect on crop yields. Can. J. Soil Sci. 40:59-62.

Martin, R.J., and R.E. Duggan. 1968. Pesticide residues in total diet samples (III). Pest. Mon. J. 1(4):11-20.

Martin, W.E., and P.R. Nickerson. 1973. Mercury, lead, cadmium, and arsenic residues in starlings - 1971. Pest. Mon. J. 7(1):67-72.

Matsumoto, N., S. Iijima, and H. Katsunuma. 1974. The burden of chemicals on the fetal body and its effect on fetal growth and development. (Transl. from Japanese.) Minzoku Eisei (People's Hygiene) 40(2):84-102.

McCarthy, D.F., and S.J. Wilson. 1957. The resistance to leaching of some water-borne preservatives in blocks of Pinus radiata when leached with distilled water. Commonwealth Scientific and Industrial Research Organization, Division of Forest Products, South Melbourne, Australia.

McCormick, E.M. 1940. The relation between the amount of arsenic a fish gets from mosquito-control dusting and the lethal dose. J. Tenn. Acad. Sci. 15(3):342-351.

Mealey, J., Jr., G.L. Brownell, and W.H. Sweet. 1959. Radioarsenic in plasma, urine, normal tissues, and intracranial neoplasms. A.M.A. Arch. Neurol. Psych. 81:310-320.

Meister, R.T., ed. 1977. Farm chemicals handbook. Meister Publishing Co., Willoughby, Ohio.

Memo. 1973. Measurement of arsenic leached from plywood, dated November 2, 1973. From Philip Haelen, Trace Elements Inc., to Glen Simon, Portland Cement Association.

Memo. 1977a. Monitoring data for dimethoate, lead arsenate (standard and basic), calcium arsenate, sodium arsenite, copper acetoarsenite (paris green), dated April 21, 1977. From F.W. Kutz, Acting Chief, Ecological Monitoring.

Memo. 1977b. Monitoring data for arsenic acid, arsenic pentoxide, sodium pyroarsenate, ammonium arsenite, arsenic trioxide, and sodium arsenate, dated July 18, 1977. From Frederick W. Kutz, Acting Chief, EMB, to William T. Waugh, Project Manager, SPRD.

Memo. 1977c. Request for usage data on inorganic arsenicals, dated August 18, 1977. From A. hale Vandermer, Program Planning & Analysis Officer, Human Effects Monitoring Branch, to William T. Waugh, Project Manager, Office of Special Pesticide Reviews.

Memo. 1978a. Confidential production data for the inorganic arsenicals, dated July 26, 1978. From Anita Schmidt, W.T. Waugh, Special Pesticide Review Division, to Edwin L. Johnson, Deputy Assistant Administrator for Pesticide Programs. CONFIDENTIAL

Menhinick, E.F. 1962. Comparison of invertebrate populations of soil and litter of mowed grasslands in areas treated and untreated with pesticides. Ecology 43(3):556-561.

Miles, J.R.W. 1968. Arsenic residues in agricultural soils of southwestern Ontario. J. Agric. Food Chem. 16(4):620-622.

Milham, S., Jr., and T. Strong. 1974. Human arsenic exposure in relation to a copper smelter. Environ. Res. 7:176-182.

Misra, S.G., and R.C. Tiwari. 1963. Studies on arsenite-arsenate system adsorption of arsenate. Soil Sci. Plant Nutr. 9(6):10-13.

Mizuta, N., M. Mizuta, F. Ito, T. Ito, H. Uchida, Y. Watanabe, H. Akama, T. Murakami, F. Hayashi, K. Nakamura, T. Yamaguchi, W. Mizuia, S. Oishi, and H. Matsumura. 1956. An outbleak [sic] of acute arsenic poisoning caused by arsenic contaminated soy-sauce (shoyu): a clinical report of 220 caces [sic]. Bull. Yamaguchi Med. School 4(2,3):131-149.

Moore, L.O. 1976. Mutagenesis and carcinogenesis tests with monosodium methanearsonate, cacodylic acid and arsenic trioxide by bacteria and cell culture tests. The Ansul Company, Weslaco, Texas.

Morris, H.E., and D.B. Swingle. 1927. Injury to growing crops caused by the application of arsenical compounds to the soil. J. Agr. Res. 34(1):59-78.

Morris, H.P., E.P. Laug, H.J. Morris, and R.L. Grant. 1938. The growth and reproduction of rats fed diets containing lead acetate and arsenic trioxide and the lead and arsenic content of newborn and suckling rats. J. Pharmacol. Exp. Therap. 64:420-445.

Muth, O.H., P.D. Whanger, P.H. Weswig, and J.E. Oldfield. 1971. Occurrence of myopathy in lambs of ewes fed added arsenic in a selenium-deficient ration. Amer. J. Vet. Res. 32(10):1621-1623.

NAS. National Academy of Sciences, Safe Drinking Water Committee. 1977a. Summary report: drinking water and health. Washington, D.C.

NAS. National Academy of Sciences, Committee on Medical and Biologic Effects of Environmental Pollutants. 1977b. Medical and biologic effects of environmental pollutants: arsenic. Washington, D.C.

NCMA. National Concrete Masonry Association. 1976. Sampling and analysis of household atmosphere; sampling and analysis of household dust. (Unpublished.)

Neal, P.A., W.C. Dreessen, T.I. Edwards, W.H. Reinhart, S.H. Webster, H.T. Castberg, and L.T. Fairhall. 1941. A study of the effect of lead arsenate exposure on orchardists and consumers of sprayed fruit. Pub. Hlth. Bulletin 267. U.S. Pub. Hlth. Ser., Federal Security Agency, Washington, D.C., 181pp.

Nelson, W.C., M.H. Lykins, J. Mackey, V.A. Newill, J.F. Finklea, and D.I. Hammer. 1973. Mortality among orchard workers exposed to lead arsenate spray: a chohort study. J. Chron. Dis. 26:105-118.

Nelson, D.A., A. Calabrese, B.A. Nelson, J.R. MacInnes, and D.R. Wenzloff. 1976. Biological effects of heavy metals on juvenile bay scallops, Argopecten irradians, in short-term exposures. Bull. Environ. Contam. Toxicol. 16(3):275-282.

Neubauer, O. 1947. Arsenical cancer: a review. Brit. J. Cancer 1:192-251.

Nielsen, F.H., S.H. Givand, and D.R. Myron. 1975. Evidence of a possible requirement for arsenic by the rat. (Abstract.) Fed. Proc. 34:923.

NIOSH. National Institute for Occupational Safety and Health. 1975. Occupational exposure to inorganic arsenic: new criteria - 1975. U.S. Dept. of Health, Education, and Welfare, Washington, D.C.

Nishioka, H. 1975. Mutagenic activities of metal compounds in bacteria. Mut. Res. 31:185-189.

Nordberg, G.F., ed. 1976. Effects and dose-response relationships of toxic metals. Elsevier Scientific Publ. Co., New York.

Oak Ridge National Laboratory, Biomedical Studies Group. 1976. Review of the environmental effects of arsenic. Oak Ridge, Tennessee. CONFIDENTIAL.

Oehme, F.W. 1972. Mechanisms of heavy metal toxicities. Clin. Toxicol. 5(2):151-167.

Ohira, M., and H. Aoyama. 1973. Epidemiological studies on the Morinaga powdered milk poisoning incident: final report of the joint project team from Hiroshima and Okayama Universities for survey of the Seno area. (Transl. from Japanese.) Jap. J. Hyg. 27(6):500-531.

Olson, O.E., L.L. Sisson, and A.L. Moxon. 1940. Absorption of selenium and arsenic by plants from soils under natural conditions. Soil Sci. 50:115-118.

Oppenheim, J.J., and W.N. Fishbein. 1965. Induction of chromosome breaks in cultured normal human leukocytes by potassium arsenite, hydroxyurea and related compounds. Cancer Res. 25:980-985.

Osburn, S.A. 1969. Lung cancer in a mining district in Rhodesia. S. Afr. Med. J. 43:1307-1312.

OSHA. U.S. Department of Labor, Occupational Safety and Health Administration. 1977. Final environmental impact statement: inorganic arsenic. Washington, D.C.

OSHA. U.S. Department of Labor, Occupational Safety and Health Administration. 1978. Occupational exposure to inorganic arsenic. Fed. Reg. 43(88):19584-19631.

Ott, G. 1976. Discussion of epidemiology of arsenic. Pages 313-314 in B.W. Carnow, ed., Health effects of occupational lead and arsenic exposure: a symposium. National Institute for Occupational Safety and Health, Washington, D.C.

Ott, M.G., B.B. Holder, and H.L. Gordon. 1974. Respiratory cancer and occupational exposure to arsenicals. Arch. Environ. Health 29:250-255.

Paton, G.R., and A.C. Allison. 1972. Chromosome damage in human cell cultures induced by metal salts. Mut. Res. 16:332-336.

PBRC. Pacific Biomedical Research Center, University of Hawaii at Manoa. Undated. Occupational and residential exposure to arsenic based wood preservatives. (Unpublished.)

Penrose, W.R. 1974. Arsenic in the marine and aquatic environments: analysis, occurrence, and significance. CRC Crit. Rev. Environ. Control 4(4):465-82.

Peoples, S.A. 1964. Arsenic toxicity in cattle. Annals N.Y. Acad. Sci. 111:644-649.

Peoples, S.A. 1975. Testimony at OSHA hearings on inorganic arsenic. Hearing transcript, pp. 1222-1239.

Petres, J., and A. Berger. 1972. The effect of inorganic arsenic on DNA synthesis of human lymphocytes in vitro. (Transl. from German.) Arch. Derm. Forsch. 242:343-352.

Petres, J., and M. Hagedorn. 1975. The "Kaiserstuhl sickness," a model of chronic arsenic intoxication. (Transl. from German.) Aktuelle Dermatol. 4(1):177-185.

Petres, J., and M. Hundeiker. 1968. "Chromosome pulverization" after arsenic action on cell cultures in vitro. (Transl. from German.) Arch. Clin. Exper. Derm. 231:366-370.

Petres, J., K. Schmidt-Ullrich, and U. Wolf. 1970. Chromosomal aberrations in human lymphocytes in cases of chronic arsenical damage. (Transl. from German.) Dtsch. Med. Wschr. 95:79-80.

Petres, J., D. Baron, and I. Kunick. 1974. Investigations on arsenic-caused alterations of the nucleic acid synthesis in vitro. (Transl. from German.) Dermatol. Monatsschrift 9(1):724-729.

Petres, J., D. Baron, and J. Enderle. 1975. The stimulating effect of arsenate on the incorporation of ^{14}C-thymidine into the DNA of phytohemagglutinin-treated human lymphocytes. (Transl. from German.) Arch. Derm. Forsch. 251:301-309.

Petres, J., D. Baron, and M. Hagedorn. 1977. Effects of arsenic cell metabolism and cell proliferation: cytogenetic and biochemical studies. Environ. Hlth. Perspect. 19:223-227.

Pinto, S.S., and B.M. Bennett. 1963. Effect of arsenic trioxide exposure on mortality. Arch. Environ. Health 7:583-591.

Pinto, S.S., M.O. Varner, K.W. Nelson, A.L. Labbe, and L.D. White. 1976. Arsenic trioxide absorption and excretion in industry. J. Occup. Med. 18(10):677-680.

Pinto, S.S., P.E. Enterline, V. Henderson, and M.O. Varner. 1977. Mortality experience in relation to a measured arsenic trioxide exposure. Environ. Health Perspec. 19:127-130.

Porter, E.K., and P.J. Peterson. 1975. Arsenic accumulation by plants on mine waste (United Kingdom). Sci. Total Environ. (Netherlands). 4:365-371.

Puzanova, L., and M. Doskocil. 1976. Mechanism of the development of malformations in chick embryos after administration of arsenic. (Transl. from Czech.) Sbornik Lekarsky 78:57-63.

Ratsch, H.C. 1974. Heavy-metal accumulation in soil and vegetation from smelter emissions. National Ecological Research Center, Environmental Protection Agency, Corvallis, Oregon. Report No. EPA-660/3-74-012, 23 pp.

Rentoul, E., and H. Smith., eds. 1973. Arsenic and its compounds. Pages 537-548 in Glaister's medical jurisprudence and toxicology, 13th ed. Churchill Livingstone, London.

Ridgway, L.P., and D.A. Karnofsky. 1952. The effects of metals on the chick embryo: toxicity and production of abnormalities in development. Ann. N.Y. Acad. Sci. 55:203-215.

Romanoff, A.L., and A.J. Romanoff, eds. 1949. The avian egg. J. Wiley & Son, New York.

Rossman, T.G., M.S. Meyn, and W. Troll. 1975. Effects of sodium arsenite on the survival of UV-irradiated Escherichia coli: inhibition of a rec A-dependent function. Mut. Res. 30:157-162.

Rossman, T.G., M.S. Meyn, and W. Troll. 1977. Effects of arsenite on DNA repair in Escherichia coli. Environ. Health Persp. 19:229-233.

Roth, F. 1956. Concerning chronic arsenic poisoning of the Moselle wine growers with special emphasis on arsenic carcinoma. (Transl. from German.) Z. f. Krebsforschung 61:287-319.

Roth, F. 1957a. Concerning the delayed effects of the chronic arsenism of the Moselle wine growers. (Transl. from German.) Dtsch. Med. Wschr. 82:211-217.

Roth, F. 1957b. The sequelae of chronic arsenic poisoning in Moselle vintners. Germ. Med. Monthly. 2(6):172-175.

Roth, F. 1957c. Arsenic-liver-tumors: hemangioendothelioma. (Transl. from German.) Z. Krebsforsch 61:468-503.

Roth, F. 1958. Concerning the bronchial cancers of arsenic-impaired wine growers. (Transl. from German.) Virchow's Archiv 331:119-137.

Rothman, S. 1954. Physiology and biochemistry of the skin. The University of Chicago Press, Chicago. 741 pp.

Rothstein, A. 1963. Interactions of arsenate with the phosphate-transporting system of yeast. J. Gen. Physiol. 46:1075-1085.

Rumburg, C.B., R.E. Engel, and W.F. Meggitt. 1960. Effect of phosphorus concentration on the absorption of arsenate by oats from nutrient solution. Agron. J. 52:452-453.

Sachs, R.M., and J.L. Michael. 1971. Comparative phytotoxicity among four arsenical herbicides. Weed Sci. 19(5):558-564.

Sandberg, G.R., and I.K. Allen. 1975. A proposed arsenic cycle in an agronomic ecosystem. Pages 124-147 in E.A. Woolson, ed., Arsenical pesticides. ACS Symposium Series 7. American Chemical Society, Washington, D.C.

Savchuck, W.B., H.W. Loy, and S.S. Schiaffino. 1960. Effect of arsenic on growth of mammalian cells in vitro. Proc. Soc. Exp. Biol. Med. 105:543-547.

Sawada, N., and L.I. Rebhun. 1969. The effect of dinitrophenol and other phosphorylation uncouplers on the birefringence of the mitotic apparatus of marine eggs. Exper. Cell Res. 55:33-38.

Schroeder, H.A., and J.J. Balassa. 1966. Abnormal trace metals in man: arsenic. J. Chronic Dis. 19:85-106.

Schroeder, H.A., and M. Mitchener. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch. Environ. Health 23:102-106.

Schuth, C.K., A.R. Isensee, E.A. Woolson, and P.C. Kearney. 1974. Distribution of ^{14}C and arsenic derived from ^{14}C cacodylic acid in an aquatic ecosystem. J. Agr. Food Chem. 22(6):999-1003.

Skipper, H.E., J.H. Mitchell, Jr., L.L. Bennett, Jr., M.A. Newton, L. Simpson, and M. Eidson. 1951. Observations on inhibition of nucleic acid synthesis resulting from administration of nitrogen mustard, urethan, colchicine, 2,6-diaminopurine, 8-azaguanine, potassium arsenite, and cortisone. Cancer Res. 11:145-149.

Sleater, G.S., and H.W. Berger. 1977. Final report on study of arsenic in selected all weather wood foundations (AWWF). Department of Housing & Urban Development, Washington, D.C. (Unpublished.)

Small, H.G., Jr., and C.B. McCants. 1962. Influence of arsenic applied to the growth media on the arsenic content of flue-cured tobacco. Agron. J. 54:129-133.

Smith, T.J., D.J. Eatough, L.D. Hansen, and N.F. Mangelsen. 1976. The chemistry of sulfur and arsenic in airborne copper smelter particulates. Bull. Environ. Contam. Toxicol. 15(6):651-659.

Sohacki, L.P. 1968. Dynamics of arsenic in the aquatic environment. Ph.D. Thesis. Michigan State University, Ann Arbor, Michigan, 101 pp.

Sorensen, E.M.B. 1976a. Toxicity and accumulation of arsenic in green sunfish, Lepomis cyanellus, exposed to arsenate in water. Bull. Environ. Contam. Toxicol. 15(6): 756-761.

Sorensen, E.M.B. 1976b. Thermal effects on the accumulation of arsenic in green sunfish, Lepomis cyanellus. Arch. Environ. Contam. Toxicol. 4:8-17.

Sram, R.J., and V. Bencko. 1974. Contributory article on evaluation of the genetic risk of exposure to arsenic. (Transl. from Russian.) Ceskoslovenska Hygiena (Prague) 19(6-7):308-315.

Steevens, D.R., L.M. Walsh, and D.R. Keeney. 1972a. Arsenic residues in soil and potatoes from Wisconsin potato fields - 1970. Pest. Mon. J. 6(2):89-90.

Steevens, D.R., L.M. Walsh, and D.R. Keeney. 1972b. Arsenic phytotoxicity on a Plainfield sand as affected by ferric sulfate or aluminum sulfate. J. Environ. Qual. 1(3):301-303.

Stellman, J.M., and G. Kabat. 1978. An assessment of the health effects of arsenic (draft). U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C.

Stellwaag, F. 1928. Viticulture insects in grape growing regions: text and handbook. (Transl. from German.) Paul Parey, Berlin.

Stellwaag, F. 1936. Pest control in viniculture. (Transl. from German.) Found. Adv. Horti- Vinicult. 24:29-57.

Stewart, J., and E.S. Smith. 1922. Some relations of arsenic to plant growth: part 2. Soil Sci. 14:119-126.

Sullivan, R.J. 1969. Air pollution aspects of arsenic and its compounds. U.S. Dept. of Health, Education, and Welfare, Natl. Air Pollution Control Adm.

Surber, E.W., and O.L. Meehan. 1931. Lethal concentrations of arsenic for certain aquatic organisms. Trans. Am. Fish. Soc. 61:225-239.

Suta, B.E. 1978. Human exposures to atmospheric arsenic. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C.

Swiggart, R.C., C.J. Whitehead, Jr., A. Curley, and F.E. Kellogg. 1972. Wildlife kill resulting from the misuse of arsenic acid herbicide. Bull. Environ. Contam. Toxicol. 8(2):122-128.

Tammes, P.M., and M.M. deLint. 1969. Leaching of arsenic from soil. Neth. J. Agric. Sci. 17:128-132.

Thiers, H., D. Colomb, G. Moulin, and L. Colin. 1967. Arsenical cutaneous cancer of the vinegrowers of Beaujolais. (Transl. from French.) Ann. Dermatol. Syph., Paris 94(2): 133-158.

Thuret, M.J. 1939. The transformation of arsenical compounds into arsenous oxide in the hepatic cell. (Transl. from French.) J. Pharm. Chimie 29:5-11.

Tokudome, S., and M. Kuratsune. 1976. A cohort study on mortality from cancer and other causes among workers at a metal refinery. Int. J. Cancer 17:310-317.

Tseng, W-P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspec. 19:109-119.

Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40(3):453-463.

Tsuda, S. 1974. Effects of 2,4-dinitrophenol, sodium arsenate, and oligomycin on nitosis of mouse L cells growing in monolayer culture. Tokushima J. Exp. Med. 21:49-59.

Ullmann, W.W., R.W. Schaefer, and W.W. Sanderson. 1961. Arsenic accumulation by fish in lakes treated with sodium arsenite. J. Water Pollut. Contr. Fed. 33(4):416-418.

USDA. U.S. Department of Agriculture. 1967. PR Notice 67-2. Loose-leaf pub. 1 p.

USDA. U.S. Department of Agriculture. 1968a. PR Notice 68-3. Loose-leaf pub. 5 pp.

USDA. U.S. Department of Agriculture. 1968b. PR Notice 68-8. Loose-leaf pub. 5 pp.

USDA. U.S. Department of Agriculture. 1968c. PR Notice 68-19. Loose-leaf pub. 2 pp.

USDA. U.S. Department of Agriculture. 1968d. PR Notice 68-5. Loose-leaf pub. 8 pp.

USDA. U.S. Department of Agriculture. 1968e. PR Notice 68-9. Loose-leaf pub. 5 pp.

USDA. U.S. Department of Agriculture. 1968f. PR Notice 68-15. Loose-leaf pub. 2 pp.

USDA. U.S. Department of Agriculture. 1968g. PR Notice 68-6. Loose-leaf pub. 14 pp.

USDA. U.S. Department of Agriculture. 1969a. PR Notice 69-1. Loose-leaf pub. 6 pp.

USDA. U.S. Department of Agriculture. 1969b. PR Notice 69-3. Loose-leaf pub. 3 pp.

USDA. U.S. Department of Agriculture. 1969c. PR Notice 69-20. Loose-leaf pub. 3 pp.

USDA. U.S. Department of Agriculture. 1969d. PR Notice 69-4. Loose-leaf pub. 8 pp.

USDA. U.S. Department of Agriculture. 1970a. PR Notice 70-1. Loose-leaf pub. 5 pp.

USDA. U.S. Department of Agriculture. 1970b. PR Notice 70-4. Loose-leaf pub. 7 pp.

Vandecaveye, S.C. 1943. Growth and composition of crops in relation to arsenical spray residues in the soil. Proc. 6th Pacific Sci. Cong. Pacific Sci. Assn. 6:217-223.

Vandecaveye, S.C., G.M. Horner, C.M. Keaton. 1936. Unproductiveness of certain orchard soils as related to lead arsenate spray accumulations. Soil Sci. 42:203-213.

van Emden, F. 1936. Animal pests damaging vines, currants, gooseberries and strawberries. (Transl. from German.) Leipzig.

von Pein, H. 1943. Carcinogenesis in chronic arsenic poisoning. (Transl. from German.) Arch. f. Klin. Med. 190:429-443.

Walsh, L.M., and D.R. Keeney. 1975. Behavior and phytotoxicity of inorganic arsenicals in soils. Pages 35-51 in E.A. Woolson, ed., Arsenical pesticides. ACS Symposium Series 7. American Chemical Society, Washington, D.C.

Walsh, D.F., B.L. Berger, and J.R. Bean. 1977. Mercury, arsenic, lead, cadmium, and selenium residues in fish, 1971-73 - National Pesticide Monitoring Program. Pest. Mon. J. 11(1):5-34.

Watanabe, T., Y. Fujimoto, and H. Nakamura. 1971. Absorption of arsenic and lead by eggplant from soil and their translocation. Bull. Agr. Chem. Inspect. Stn. (Tokyo) 11:101-105.

Weast, R.C., ed. 1977. Handbook of chemistry and physics, 58th ed. C.R.C. Press, Inc., Cleveland, Ohio.

Webb, J.L. 1966. Enzyme and metabolic inhibitors. Vol. III. iodoacetate, maleate, N-ethylmaleimide, alloxan, quinones, arsenicals. Academic Press, New York.

Webster, S.H. 1941. Lead and arsenic ingestion and excretion in man. Pub. Health Rpts. 56(2):1359-1368.

Weir, P.A., and C.H. Hine. 1970. Effects of various metals on behavior of conditioned goldfish. Arch. Environ. Health 20:45-51.

White, D.H., J.R. Bean, and J.R. Longcore. 1977. Nationwide residues of mercury, lead, cadmium, arsenic, and selenium in starlings, 1973. Pest. Mon. J. 11(1):35-39.

WHO. World Health Organization. 1973. Hazards to health of persistant substances - arsenic (draft).

Wiebe, A.H. 1930. Notes on the exposure of young fish to varying concentrations of arsenic. Trans. Am. Fish. Soc. 60:270-278.

Williams, K.T., and R.R. Whetstone. 1940. Arsenic distribution in soils and its presence in certain plants. Technical Bulletin 732. U.S. Government Printing Office, Washington, D.C.

Winkler, W.O. 1962. Identification and estimation of the arsenic residue in livers of rats ingesting arsenicals. J.A.O.A.C. 45(1):80-91.

Wolfe, H.R., W.F. Durham, and J.F. Armstrong. 1967. Exposure of workers to pesticides. Arch. Environ. Health 14:622-632.

Woolson, E.A. 1972. Effects of fertiliser materials and combinations on the phytotoxicity, availability and content of arsenic in corn (maize). J. Sci. Food Agr. 23:1477-1481.

Woolson, E.A. 1973. Arsenic phytotoxicity and uptake in six vegetable crops. Weed Sci. 21(6):524-527.

Woolson, E.A. 1975. Bioaccumulation of arsenicals. Pages 97-107 in E.A. Woolson, ed., Arsenical pesticides. ACS Symposium Series 7. American Chemical Society, Washington, D.C.

Woolson, E.A. 1976. Transport, transformation and fate of arsenic in aquatic ecosystems and soils. U.S. Department of Agriculture, Agricultural Research Service, Beltsville, Maryland. (Unpublished.)

Woolson, E.A., and P.C. Kearney. 1973. Persistence and reactions of ^{14}C-cacodylic acid in soils. Environ. Sci. Technol. 7(1):47-50.

Woolson, E.A., J.H. Axley, and P.C. Kearney. 1971a. Correlation between available soil arsenic, estimated by six methods, and response of corn (*Zea mays* L.). Soil Sci. Amer. Proc. 35:101-105.

Woolson, E.A., J.H. Axley, and P.C. Kearney. 1971b. The chemistry and phytotoxicity of arsenic in soils. I. contaminated field soils. Soil Sci. Soc. Amer. Proc. 35:938-943.

Yeh, S. 1963. Relative incidence of skin cancer in Chinese in Taiwan: with special reference to arsenical cancer. Natl. Cancer Inst. Monogr. 10:81-107.

Yeh, S. 1973. Skin cancer in chronic arsenicism. Hum. Pathol. 4(4):469-485.

Yeh, S., S.W. How, and C.S. Lin. 1968. Arsenical cancer of skin: histologic study with special reference to Bowen's disease. Cancer 21(2):312-339.

**** PRODUCT SEARCH LISTING **** PAGE 1

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* C00004 BONIDE CHEMICAL CO. INC.
2 WURZ AVE.
YORKVILLE NY 13495

********** PRODUCT NAME **********

**00109* BONIDE SODIUM ARSENITE GRASS & WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000005 EMPIRE INTERNATIONAL
P.O. BOX 29805
ATLANTA, GA 30359

********** PRODUCT NAME **********

**00031* EM 1400 (SODIUM ARSENITE SOLUTION) NON-SELECTIVE WEED & BRUSH KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000029 JONES PRODUCT COMPANY
BOX 204
MIDDLETON, WI 53562

********** PRODUCT NAME **********

**00004* JONES ANT KILLER (NEW SQUEEZE CAN ANT KILLER)

**** PRODUCT SEARCH LISTING **** PAGE 2

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000030 SWEENEY W R MFR INC
340 S MAIN ST
SALISBURY MO 65281

********** PRODUCT NAME **********

**00001* SWEENEY'S ANT-GO

****

REGISTRANT *NAME AND ADDRESS*

* 000055 FATSCO
251 N FAIR AVE
BENTON HARBOR MI 49022

********** PRODUCT NAME **********

**00001* FATSCO ANT POISON FOR SWEET EATING ANTS

****

REGISTRANT *NAME AND ADDRESS*

* 000061 KOPPERS COMPANY, INCORPORATED
GENERAL OFFICES-KOOPERS BLD
PITTSBURGH PA 15219

********** PRODUCT NAME **********

**00110* WOLMAN SALTS FCAP, WOOD PRESERVATIVE

**00111* KOPPERS WOLMAN WOOD PRESERVATIVE SOLUTION

**00124* KOPPERS WOLMAN SALTS FMP

**00127* WOLMAN SALTS CCA TYPE A

**00128* WOLMANAC CONCENTRATE 50% (INDUSTRIAL USE)

**** PRODUCT SEARCH LISTING **** PAGE 3

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

**CONTINUE REGISTRANT 000061

**00134* KOPPERS MINASAL MINE TIMBER PRESERVATIVE

**00139* WOLMAN SALTS CCA-TYPE B

**00140* WOOD PRESERVATIVE CCA-TYPE C

**00141* WOLMANAC CONCENTRATE 70% FOR INDUSTRIAL USE ONLY

**00170* ALLIED CHEMICAL CCA TYPE B WOOD PRESERVATIVE

**00171* ALLIED CHEMICAL ARSENIC ACID 75%

****

REGISTRANT *NAME AND ADDRESS*

* 000106 BRULIN & COMPANY INC
PO BOX 270-B
INDIANAPOLIS IN 46206

********** PRODUCT NAME **********

**00001* BRULIN'S NON-SELECTIVE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000148 THOMPSON-HAYWARD CHEMICAL COMPANY
BOX 2383
KANSAS CITY KS 66110

********** PRODUCT NAME **********

**00674* DE PESTER DESICCANT-A

**** PRODUCT SEARCH LISTING **** PAGE 4

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000149 SENCRET CHEMICAL COMPANY
631 LEFFINGWELL AVE
ST LOUIS MO 63122

********** PRODUCT NAME **********

**00002* TERRO ANT KILLER

**00004* TERRO CALIFORNIA ANT KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000192 DEXOL INDUSTRIES
1450 W 228TH ST
TORRANCE CA 90501

********** PRODUCT NAME **********

**00095* DEXOL WEEDSOL NON SELECTIVE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000218 ALLIED CHEMICAL CORPORATION
P.O. BOX 1058-R
MORRISTOWN NJ 07960

********** PRODUCT NAME **********

**00445* ARCADIAN KILMAG

**00661* ALLIED CHEMICAL SODIUM ARSENATE

**** PRODUCT SEARCH LISTING **** PAGE 5

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000239 CHEVRON CHEMICAL COMPANY
ORTHO DIVISION 940 HENSLEY WAY
RICHMOND CA 94801

********** PRODUCT NAME **********

**00023* ORTHO BUG-GETA PELLETS

**00061* SODITE GRAPE SPRAY

**00074* METAG AGRICULTURAL BAIT (PELLETS)

**00087* ORTHO BASIC LEAD ARSENATE (PROD. 119)

**00111* ORTHO BUG-GETA MEAL

**00289* ORTHO ARSENICAL WEED KILLER

**00561* METAG-XX AGRICULTURAL BAIT(PELLETS)

**02207* (ORTHO) CHEVRON ARSENIC ACID

****

REGISTRANT *NAME AND ADDRESS*

* 000255 AMERICAN FLUORIDE CORP
17 HUNTINGTON PLACE
NEW ROCHELLE NY 10801

********** PRODUCT NAME **********

**00025* AFC ARSENIOUS OXIDE

**** PRODUCT SEARCH LISTING **** PAGE 6

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INCRG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000279 FMC CORP.
AGRICULTURAL CHEM DIV.
2000 MARKET STREET
PHILADELPHIA, PA. 19103

********** PRODUCT NAME **********

**00046* NIAGARA BASIC POWDERED ARSENATE OF LEAD

**00153* NIAGARA NEW CUCURIBIT DUST

**00825* NIAGARA 7-20 DUST FUNGICIDE-INSECTICIDE

**C1874* NIAGARA BLUEBERRY MAGGOT DUST

****

REGISTRANT *NAME AND ADDRESS*

* 000295 COMMERCIAL CHEMICAL COMPANY
P O BOX 7275
MEMPHIS, TN 38107

********** PRODUCT NAME **********

**00005* DELTA BRAND ARSENIC ACID

**00006* DELTA BRAND ARSENIC ACID F/BERMUDA GRASS,F/COMPECI OF DALLIS & CRAB

****

REGISTRANT *NAME AND ADDRESS*

* 000305 BADGER PHARMACAL INC.
P.O. BOX 171
CEDARBURG WI. 53012

********** PRODUCT NAME **********

**00006* ANT EXIT

**** PRODUCT SEARCH LISTING **** PAGE 7

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000328 GETEM MANUFACTURING CO., INC.
1261 W. 42ND STREET
NORFOLK, VA. 23508

********** PRODUCT NAME **********

**00002* GETEM ANT POISON

****

REGISTRANT *NAME AND ADDRESS*

* 000359 RHODIA INC. AGRICULTURAL DIVISION
P.O. BOX 125
MONMOUTH JUNCTION, NJ 08852

********** PRODUCT NAME **********

**00001* ATLAS "A" A SODIUM ARSENITE SOLUTION

**00228* ATLAS "A6" SODIUM ARSENITE SOLUTION

**00360* CHIP-CAL GRANULAR

**00536* CHIPMAN BETA A PELLET

****

REGISTRANT *NAME AND ADDRESS*

* 000363 COOPERS CREEK CHEMICAL CORP
RIVER ROAD
WEST CONSHOHOCKEN, PA 19428

********** PRODUCT NAME **********

**00001* MAIN LINE WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 8

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000390 PETTIT PAINT COMPANY INC
36 PINE ST
BOROUGH OF ROCKAWAY NJ 07866

********** PRODUCT NAME **********

**00011* PETTIT TROPICOP THE SUPREME PLASTIC COPPER PAINT 1342 GREEN

**00038* PETTIT MARINE PAINT ANTI-FOULING 1330 ALUMACIDE GREEN

****

REGISTRANT *NAME AND ADDRESS*

* 000402 HILL MANUFACTURING COMPANY
1500 JONESBORO RD SE
ATLANTA GA 30315

********** PRODUCT NAME **********

**00053* HILL'S NO. 572 HERBICIDE CONCENTRATE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000419 CENCL COMPANY
DIV OF BURGESS VIEBOCHARTERS INC
P O BOX 177 ROUTE 45 & PETERSON RD
LIBERTYVILLE, IL 60048

********** PRODUCT NAME **********

**00004* 7-11 ANT POISON

**** PRODUCT SEARCH LISTING **** PAGE 9

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000421 VARIEY JAMES & SONS INC
120C SWITZER AVE
ST LOUIS MO 63147

********** PRODUCT NAME **********

**00368* VARCO FOUR PLUS FIFTY NON-SELECTIVE CONCENTRATE

**00398* VARCO SIX PLUS SIXTY NON SELECTIVE CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 000422 BLUE BALL CHEMICAL COMPANY
914 HOFFNER AVENUE
ORLANDO FL 32809

********** PRODUCT NAME **********

**00017* MOL-EX

****

REGISTRANT *NAME AND ADDRESS*

* 000446 GOOD JAMES COMPANY
2107-15 E SUSQUEHANNA AVE
PHILADELPHIA PA 19125

********** PRODUCT NAME **********

**00001* WEEDEX SODIUM METARSENITE 41%

**** PRODUCT SEARCH LISTING **** PAGE 10

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000475 BOYLE-MIDWAY INC
S AVE & HALE ST
CRANFORD NJ 07016

********** PRODUCT NAME **********

**00007* ANTROL ANT KILLER

**00013* ANTROL ANT SYRUP

****

REGISTRANT *NAME AND ADDRESS*

* 000476 STAUFFER CHEMICAL COMPANY LABELING & REGISTRATION
DEPT 1200 SOUTH 47TH ST
RICHMOND, CA 94804

********** PRODUCT NAME **********

**01092* CAL-META SNAIL PELLETS

**01551* CALCIUM ARSENATE-METALDEHYDE 5-3 PELLETS

****

REGISTRANT *NAME AND ADDRESS*

* 000477 GRO ALL INC
DIV OF CENTRAL CHEM CORP
P. O. BOX 918
HAGERSTOWN, MD 21740

********** PRODUCT NAME **********

**00073* FARMRITE COP-CAL VEGETABLE DUST

**** PRODUCT SEARCH LISTING **** PAGE 11

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000483 PACIFIC SUPPLY CORP
PO BOX 3588
PORTLAND OR 97208

********** PRODUCT NAME **********

**00113* PACIFIC COOPERATIVES SLUG PELLETS

****

REGISTRANT *NAME AND ADDRESS*

* 000491 SELIG CHEMICAL INDUSTRIES THE
DIV NATIONAL SERVICE INDS INC PO BOX 43106
ATLANTA GA 30336

********** PRODUCT NAME **********

**00003* SELIG'S WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000498 CHASE PRODUCTS COMPANY
19TH ST & GARDNER BLVD
BROADVIEW IL 60155

********** PRODUCT NAME **********

**00001* CHASES'S ANT KILLER

**** PRODUCT SEARCH LISTING **** PAGE 12

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000505 COWLEY S L & SONS MFG INC
BOX 454
HUGO OK 74743

********** PRODUCT NAME **********

**00001* COWLEY'S 'ORIGINAL' RAT AND MOUSE POISON

****

REGISTRANT *NAME AND ADDRESS*

* 000551 BAIRD & MCGUIRE INC
SOUTH STREET
HOLBROOK,MA 02343

********** PRODUCT NAME **********

**00001* WEED KILLER NO. 50

**C0088* WEED KILLER NO. 50A

**00208* SODIUM ARSENITE "8"

**00214* SODIUM ARSENITE 4

****

REGISTRANT *NAME AND ADDRESS*

* C00604 HAMMOND PAINT & CHEM COMPANY INC
BOX 292
WESTWOOD MA 02090

********** PRODUCT NAME **********

**00017* HAMMOND'S WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 13

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INCRG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000677 DIAMOND SHAMROCK CORP. AGRICULTURAL CHEM. DIVISION
A UNIT OF DIAMOND SHAMROCK CORP 1100 SUPERIOR AVE
CLEVELAND OH 44114

********** PRODUCT NAME **********

**00242* SWEDISH REFINED WHITE ARSENIC POWDER

****

REGISTRANT *NAME AND ADDRESS*

* 000690 PERK PRODUCTS CO
1336 LEWIS ST
NASEVILLE,TN 37210

********** PRODUCT NAME **********

**00029* PERKERSON'S POLE-END

****

REGISTRANT *NAME AND ADDRESS*

* 000728 SOUTHLAND PEARSON & CO
PO BOX 7151
MOBILE,AL 36607

********** PRODUCT NAME **********

**00023* PEARSON'S QUIK-KILL BAIT

**** PRODUCT SEARCH LISTING **** PAGE 14

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000769 WOOLFOLK CHEMICAL WORKS INC
PO BOX 538
FT VALLEY GA 31030

********** PRODUCT NAME **********

**00014* SECURITY BRAND CALCIUM ARSENATE

**00149* SECURITY BRAND TOMATO DUST

**00287* 40% SOLUTION SODIUM ARSENITE

**00374* SECURITY FLAG

**00443* SECURITY CALNATE

****

REGISTRANT *NAME AND ADDRESS*

* 000779 RAESY & BESTHOFF INC
143 RIVER RD
EDGEWATER NJ 07020

********** PRODUCT NAME **********

**00002* WEED KILLER (SODIUM ARSENITE SOLUTION)

****

REGISTRANT *NAME AND ADDRESS*

* 000839 BELL CHEMICAL COMPANY
BOX 10007
DALLAS TX 75207

********** PRODUCT NAME **********

**00059* BELCO NON-SELECTIVE WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 15

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000842 ROBINS G S COMPANY
126 CHOUTEAU AVE
ST LOUIS MO 63102

********** PRODUCT NAME **********

**00113* SODIUM ARSENITE 6-LB. SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 000962 LOS ANGELES CHEMICAL COMPANY
4545 ARLINE ST
SOUTH GATE CA 90280

********** PRODUCT NAME **********

**00093* LACCO BRAND CALCIUM ARSENATE

**00300* LACCO BASIC LEAD ARSENATE

**00340* LACCO WHITE ARSENIC

**00344* LACCO PARIS GREEN

**00349* LACCO SODIUM ARSENITE SOLUTION NO. 6

****

REGISTRANT *NAME AND ADDRESS*

* 001022 CHAPMAN CHEMICAL COMPANY
BOX 9158
MEMPHIS TN 38109

********** PRODUCT NAME **********

**00109* NARSITE 40

**** PRODUCT SEARCH LISTING **** PAGE 16

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

**CONTINUE REGISTRANT 001022

**00414* CHAPMAN NARSITE 60

****

REGISTRANT *NAME AND ADDRESS*

* 001057 C B DOLGE COMPANY
11 PERRY LANE WEST
WESTPORT, CT 06880

********** PRODUCT NAME **********

**00020* DOLGE SUPER STRENGTH WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001266 WALTER INTERNATIONAL CORPORATION
PO BOX 6099
NEW ORLEANS LA 70114

********** PRODUCT NAME **********

**00043* MWK NON-SELECTIVE WEED KILLER FOR CONT OF WEEDS, GRASS AND OTHER VEG

****

REGISTRANT *NAME AND ADDRESS*

* 001269 DEWITT CHEMICAL COMPANY
POX 343
ATLANTA GA 30301

********** PRODUCT NAME **********

**00022* DE WITT FORMULA NO 102 WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 17

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 001270 ZEP MANUF CORP
BOX 2015
ATLANTA GA 30301

********** PRODUCT NAME **********

**00042* ZEP R-59 WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001325 WEIL CHEMICAL COMPANY
219 SCOTT STREET
MEMPHIS, TN 38112

********** PRODUCT NAME **********

**00060* DE WEED CONCENTRATED KILLER OF WEEDS

****

REGISTRANT *NAME AND ADDRESS*

* 001386 UNIVERSAL COOPERATIVES INC
3001 METRO DR. SUITE 500
MINNEAPOLIS MN 55420

********** PRODUCT NAME **********

**00136* UNICO SODIUM ARSENITE WEED KILLER SOLUTION

**00447* UNICO SNAIL AND SLUG BAIT

**** PRODUCT SEARCH LISTING **** PAGE 18

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 001421 DETTELBACH CHEMICALS CORP
ATTN WARD A R 4181 PEACHTREE RD NE
ATLANTA GA 30319

********** PRODUCT NAME **********

**00074* WEED - X

****

REGISTRANT *NAME AND ADDRESS*

* 001439 BLUE SPRUCE COMPANY
1390 VALLEY ROAD
STIRLING, NJ 07980

********** PRODUCT NAME **********

**00189* ARSENIC TRIOXIDE (TECHNICAL)

**00234* CHEM SEN 56

****

REGISTRANT *NAME AND ADDRESS*

* 001624 US BORAX & CHEMICAL CORP
3075 WILSHIRE BLVD.
LOS ANGELES, CA. 60010

********** PRODUCT NAME **********

**00055* HERBICIDCX WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 19

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 001663 GRANT LAB. DIV.
LEISURE ENTERPRISE INC.
6020 ADELINE ST.
OAKLAND CA 94608

********** PRODUCT NAME **********

**00015* GRANT'S ANT CONTROL (ANT STAKES)

****

REGISTRANT *NAME AND ADDRESS*

* 001685 STATE CHEMICAL MFG CO TEE
3100 HAMILTON AVE
CLEVELAND, OHIO 44114

********** PRODUCT NAME **********

**00026* FORMULA-444 WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001691 CHEMICAL COMPOUNDING CORP
680 ELTON AVENUE
RIVERHEAD, NY 11901

********** PRODUCT NAME **********

**00016* QUIST WEED KILLER

**00085* INSECTICIDE SODIUM ARSENITE CONCENTRA E

**** PRODUCT SEARCH LISTING **** PAGE 20

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INCRG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 001769 NATIONAL CHEMSEARCH DIV
USACHEM INC
2727 CHEMSEARCH BLVD
IRVING TX 75060

********** PRODUCT NAME **********

**00037* F-40 WEED KILLER

**00070* DEEP KILL WEED KILLER

**00121* NATIONAL CHEMSEARCH DK-80 WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001926 NAVY BRAND MFG COMPANY
5111 SW AVE
ST LOUIS MO 63110

********** PRODUCT NAME **********

**00030* WK-90 CONCENTRATED NON SELECTIVE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 002155 SCHNEIDI.INC.
1429 FAIRMONT AVE N W
ATLANTA GA 30318

********** PRODUCT NAME **********

**00026* ARSENICAI WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 21

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 002169 PATTERSON CHEMICAL COMPANY INC
1400 UNION AVE
KANSAS CITY, MO 64101

********** PRODUCT NAME **********

**00027* PATTERSON'S SODIUM ARSENITE SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 002217 PBI-GORDON CORPORATION
300 SO 3RD ST
KANSAS CITY KS 66118

********** PRODUCT NAME **********

**00454* SLUG KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 002227 SINGLETARY COMPANY INC
143 S WASHINGTON ST
ROCKY MOUNT NC 27801

********** PRODUCT NAME **********

**00001* SINGLETARY'S PEST CONTROL

**** PRODUCT SEARCH LISTING **** PAGE 22

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 002270 HUGE COMPANY INC
7625 PAGE AVE
ST. LOUIS, MO 63133

********** PRODUCT NAME **********

**00684* EXCELCIDE TW-40

****

REGISTRANT *NAME AND ADDRESS*

* 002341 ROWLIN COMPANY
2727 ST LOUIS AVE
FORT WORTH TX 76112

********** PRODUCT NAME **********

**00003* KILLANT

****

REGISTRANT *NAME AND ADDRESS*

* 002342 KERR-MCGEE CHEMICAL CORP
MGR PKG & LABELING
KERR-MCGEE CENTER
OKLAHOMA CITY OK 73102

********** PRODUCT NAME **********

**00502* PASCO PARIS GREEN AEROGRAN-5

**00525* PASCO PARIS GREEN GRANULAR 7 1/2

**** PRODUCT SEARCH LISTING **** PAGE 23

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 002443 ATLANTA CHEMICAL COMPANY INC
PO BOX 237
JAMESTOWN NC 27282

********** PRODUCT NAME **********

**00001* ATLA-ANTI-ANT

****

REGISTRANT *NAME AND ADDRESS*

* 002568 JOTUN-BALTIMORE COPPER PAINT CO.
SCM CORPORATION 501 KEY HIGHWAY
BALTIMORE MD 21230

********** PRODUCT NAME **********

**00007* SEAPROOF X-254 EVERTOX GREEN COPPER ANTI-FOULING

**00037* SEAPROOF PAINT X-255 EVERTOX BLUE COPPER ANTI-FOULING

****

REGISTRANT *NAME AND ADDRESS*

* 002831 NAPASCO INTERNATIONAL, INC
PO DRAWER 1057
THIBODAUX, LA 70301

********** PRODUCT NAME **********

**00004* NAPASCO SA-40 WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 24

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 003008 OSMOSE WOOD PRES COMPANY OF AMERICAN INC
980 ELLICOTT ST
BUFFALO NY 14209

********** PRODUCT NAME **********

**00007* OSMOSALTS

**00016* OSMOSE K-33 WOOD PRESERVATIVE

**00017* COPROCHROME WOOD PRESERVATIVE

**00021* OSMOSE SPECIAL K-33 PRESERVATIVE

**00029* SODIUM ARSENATE

**00034* OSMOSE K-33(72%)WOOD PRESERVATIVE

**00035* OSMOSE K-33 (40%) WOOD PRESERVATIVE

**00036* OSMOSE K-33-C (50%) WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 003040 EDCO CHEM COMPANY INC
BOX 5877 1048 KEY RD
COLUMBIA SC 29250

********** PRODUCT NAME **********

**00038* EDCO SODIUM ARSENITE SOLUTION 15%

**00039* EDCO SODIUM ARSENTIE SOLUTION 40 (COLORED)

**** PRODUCT SEARCH LISTING **** PAGE 25

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* C03098 BAXTER J H & COMPANY
170C S EL CAMINO REAL
SAN MATEO CA 94402

********** PRODUCT NAME **********

**00015* J. H. BAXTER & CO. CHEMONITE BASE MIX

****

REGISTRANT *NAME AND ADDRESS*

* 003231 COBRA WOOD TREATMENT CORPORATION
275 MADISON AVENUE
NEW YORK NY 10016

********** PRODUCT NAME **********

**00001* COBRA SALTS

****

REGISTRANT *NAME AND ADDRESS*

* 003234 PAX CO
580 W 13TH ST SOUTH
SALT LAKE CITY UT 84115

********** PRODUCT NAME **********

**00003* PAX 400 3 YEAR CRABGRASS CONTROL

**** PRODUCT SEARCH LISTING **** PAGE 26

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 003324 GENERAL PEST SERVICE CO
2015 PONTIUS AVE
LOS ANGELES, CA 90025

********** PRODUCT NAME **********

**00003* ANT JEX REDWOOD ANT STAKES

****

REGISTRANT *NAME AND ADDRESS*

* 003840 AMERICAN FIRESTONE CORPORATION
825 THIRD AVENUE
NEW YORK NY 10022

********** PRODUCT NAME **********

**00002* REFINED WHITE ARSENIC ARSENIC TRIOXIDE (TECHNICAL)

**00005* REFINED WHITE ARSENIC ARSENIC TRIOXIDE (TECHNICAL) MINIMUM 94% AS2O3

****

REGISTRANT *NAME AND ADDRESS*

* 003862 AEC COMPOUNDING COMPANY
BOX 932
ATLANTA GA 30301

********** PRODUCT NAME **********

**00043* 40% SODIUM ARSENITE SOLUTION

**** PRODUCT SEARCH LISTING **** PAGE 27

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 004012 CHEMISTRY HALL LABORATORIES INC
BOX 1448
BRADENTON FL 33505

********** PRODUCT NAME **********

**00005* GRASS AND WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 004450 CHEMEX CHEMICALS & COATINGS CO INC
P O BOX 5072 2822 N 35TH ST
TAMPA, FL 33605

********** PRODUCT NAME **********

**00006* XD-542 NON SELECTIVE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 004581 PENNWALT CORP AGCHEM DIV.
PENNWALT TECHNOLOGICAL CENTER
P.O. BOX C
KING OF PRUSSIA, PA 19406

********** PRODUCT NAME **********

**00102* PENITE-8 SODIUM ARSENITE SOLUTION

**00205* PENITE 35

**00229* PENITE - 6

**00231* DESICCANT L-10

**00259* ARSENIC TRIOXIDE

**** PRODUCT SEARCH LISTING **** PAGE 28

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

**CONTINUE REGISTRANT C04581

**00268* SCORCH

****

REGISTRANT *NAME AND ADDRESS*

* 004715 COLORADO INTERNATIONAL CORP
7800 MARBLE N.E.
ALBUQUERQUE, N.M. 87110

********** PRODUCT NAME **********

**00122* BEST 4 SERVIS BRAND SUPER 10 ARSENIC ACID COTTON DESICCANT

****

REGISTRANT *NAME AND ADDRESS*

* 004931 GOOD-LIFE CHEMICALS INC
BOX 687
BFFINGHAM IL 62401

********** PRODUCT NAME **********

**00033* SODIUM ARSENITE 42.5% SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 004972 PROTEXALL CHEM INC
5576 S. RIDGEWOOD (HARBOR OAKS)
DAYTONA BEACH, FL 32019

********** PRODUCT NAME **********

**00008* PROTEXALL "ANT-KIL"

**** PRODUCT SEARCH LISTING **** PAGE 29

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 005535 ADINES INC J & L
182-12 93RD AVE
JAMAICA NY 11423

********** PRODUCT NAME **********

**00035* GRO-WELL PRE-VENT

****

REGISTRANT *NAME AND ADDRESS*

* 005664 CANTOL INC
2211 N AMERICAN STREET
PHILADELPHIA, PA 19133

********** PRODUCT NAME **********

**00001* SURE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 006294 COMET MANUFACTURING COMPANY
1381 DALTON DRIVE N E
ATLANTA GA 30306

********** PRODUCT NAME **********

**00022* CMC WEED KILLER(SODIUM ARSENITE)WEED-X

**** PRODUCT SEARCH LISTING **** PAGE 30

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INCRG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 006720 SOUTHERN MILL CREEK PRODUCTS COMPANY INC
BOX 1096
TAMPA FL 33601

********** PRODUCT NAME **********

**00031* SMCP PARIS GREEN GRANULAR-7 1/2

**00070* SMCP SLUG AND SNAIL BAIT PELLETS

**00150* SMCP SODIUM ARSENITE "4" SOLUTION

00181* ********

****

REGISTRANT *NAME AND ADDRESS*

* 006762 STERN CHEM CORP
BOX 5070
MONROE LA 71201

********** PRODUCT NAME **********

**00029* S.C. WEED KILLER TYPE "B"

****

REGISTRANT *NAME AND ADDRESS*

* 006837 WILBAR INC
BOX 3111
CHARLOTTE NC 28203

********** PRODUCT NAME **********

**00022* WII-KILL ARSENICAL WEED KILLER

**C0030* SURE-KILL

**** PRODUCT SEARCH LISTING **** PAGE 31

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INCRG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 007001 OCCIDENTAL CHEMICAL CO
P O BOX 198
LATHROP, CA 95330

********** PRODUCT NAME **********

**00141* BEST SNAIL & SLUG PELLETS DOUBLE 5

****

REGISTRANT *NAME AND ADDRESS*

* 007273 CROWN CHEMICAL INCORPORATED
4995 NORTH MAIN STREET
ROCKFORD IL 61101

********** PRODUCT NAME **********

**00061* FLORATOX 40 TREE KILLER AND CONTACT WEED KILLER

**00072* FLORA-TOX 60 TREE KILLER AND CONTACT WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 007401 VOLUNTARY PURCHASING GROUP INC
PO BOX 460
ROMEAN TX 75418

********** PRODUCT NAME **********

**00005* FERTI-LOME WEED & WILD GRASS KILLER

**00184* POLY BRAND DESICCANT

**00186* FERTI LOME DAL GRAB

**00195* HI-YIELD H-10 CONTAINING T-45

**** PRODUCT SEARCH LISTING **** PAGE 32

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

**CONTINUE REGISTRANT 007401

**00200* HI-YIELD SINERGIZED H-10

**00202* REFINED WHITE ARSENIC

**00204* HI-YIELD GENERAL WEED KILLER

**00205* HI-YIELD DECIMEX SURFACTANT

****

REGISTRANT *NAME AND ADDRESS*

* 007992 T N T CHEMICALS INC
7301 NW 77TH ST
MIAMI FL 33166

********** PRODUCT NAME **********

**00005* TRULY NOIEN TREATMENT TNT ANT FREE

****

REGISTRANT *NAME AND ADDRESS*

* 008047 POLY CHEM INC
P. O. BOX 10026
NEW ORLEANS, LA 70121

********** PRODUCT NAME **********

**00009* POLY WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 33

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INCRG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 008333 TACC CORPORATION
BOX 2909
SPARTANBURG SC 29302

********** PRODUCT NAME **********

**00001* BOLIDEN-CCA

**00002* TACO CCA CONC 40%

****

REGISTRANT *NAME AND ADDRESS*

* 009777 AMETALCO INC
530 5TH AVE
NEW YORK NY 10036

********** PRODUCT NAME **********

**00001* ARSENIC TRIOXIDE

****

REGISTRANT *NAME AND ADDRESS*

* C09791 YUKON SERVICE COMPANY
30 EAST 42ND STREET
NEW YORK NY 10017

********** PRODUCT NAME **********

**00004* 6840-281-2030 INSECT. SOD.ARSENITE CONC FED. SPECIF. O-I-579

**** PRODUCT SEARCH LISTING **** PAGE 34

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 009881 CHILTON PAINT COMPANY
109-09 15 AVE
COLLEGE POINT NY 11356

********** PRODUCT NAME **********

**00001* CHILTON SUPER DRIPLESS #8 BOTTOM PAINT

**00002* CHILTON DRIPLESS BOTTOM PAINT RED

**00003* CHILTON METALLIC COPPER PAINT ORIGINAL FORMULA NO. 888 RED

**00004* CHILTON METALLIC COPPER PAINT NEW DRIPLESS BLUE

****

REGISTRANT *NAME AND ADDRESS*

* 010193 SHEEP CHEMICAL & SUPPLY COMPANY
2827 RIVERVIEW BLVD
BRADENTON FL 33505

********** PRODUCT NAME **********

**00001* NO-GRO LIQUID CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 010204 MARKO CHEM INC
429 ALEXANDER AVE.
SPARTANBURG SC 29302

********** PRODUCT NAME **********

**00011* MARKO KILL-KO ARSENICAL WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 35

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 010465 MINERAL RESEARCH & DEVELOPMENT CORP
BOX 9111
CHARLOTTE NC 28201

********** PRODUCT NAME **********

**C0010* CCA TYPE C WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011656 WESTERN FARM SERVICE INC SHELL CHEM COMPANY
1025 CONNECTICUT AVE-STE 200
WASH DC 20036

********** PRODUCT NAME **********

**C0022* COASTOX META CAL 3=5 PELLETS

****

REGISTRANT *NAME AND ADDRESS*

* 015135 UNITED MINERAL & CHEMICAL CORP
129 HUDSON ST
NEW YORK NY 10013

********** PRODUCT NAME **********

**00001* SODIUM ARSENATE (IMPORT)

**** PRODUCT SEARCH LISTING **** PAGE 36

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* C20004 TRAYLOR CHEMICAL & SUPPLY COMPANY
PO BOX 7937 1911 LOCKWOOD WAY
ORLANDO FL 32804

********** PRODUCT NAME **********

**00003* TRACO ARSENIC ACID

****

REGISTRANT *NAME AND ADDRESS*

* 022058 SHARP CHEMICAL COMPANY
5921 PLAINVIEW
HOUSTON TX 77017

********** PRODUCT NAME **********

**00002* SHARP SODIUM ARSENATE SOLUTION 40 PERCENT

****

REGISTRANT *NAME AND ADDRESS*

* 035896 CP CHEMICALS, INC.
ABROR STREET
SEWAREN, NJ 07077

********** PRODUCT NAME **********

**00002* WOOD-LAST CONC. WOOD PRESERV. AQ.50% SOL.CCA-TYPE A

**** PRODUCT SEARCH LISTING **** PAGE 37

08/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 040655 RBS DISTRIBUTORS INC.
1816 CORTEZ RD WEST APT. 102A
BRADENTON FL. 33507

********** PRODUCT NAME **********

**00001* TEX ANT ANT KILL

**** PRODUCT SEARCH LISTING **** PAGE 1

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000004 BONIDE CHEMICAL CO. INC.
2 WURZ AVE.
YORKVILLE NY 13495

********** PRODUCT NAME **********

**03823* BONIDE MOLETOX POISON PEANUTS

****

REGISTRANT *NAME AND ADDRESS*

* 000239 CHEVRON CHEMICAL COMPANY
ORTHO DIVISION 940 BENSLEY WAY
RICHMOND CA 94801

********** PRODUCT NAME **********

**04254* ORTHO STANDARD LEAD ARSENATE

****

REGISTRANT *NAME AND ADDRESS*

* 000422 BLUE BALL CHEMICAL COMPANY
914 HOFFNER AVENUE
ORLANDO FL 32809

********** PRODUCT NAME **********

**05374* BLUE BALL ANT KILLER

**05375* MOL-EX

**05379* BLUE BALL RAT KILLER

**** PRODUCT SEARCH LISTING **** PAGE 2

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 000850 BONIED CHEMS CORP
689 S MAIN ST
LIMA OH 45801

********** PRODUCT NAME **********

**10203* KILL-ALL CONCENTRATED WEED & GRASS KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000962 LOS ANGELES CHEMICAL COMPANY
4545 ARLINE ST
SOUTH GATE CA 90280

********** PRODUCT NAME **********

**10173* SODIUM ARSENITE SOLUTION NO.4

****

REGISTRANT *NAME AND ADDRESS*

* 005905 HELENA CHEMICAL CO
CLARK TOWER, 5100 POPLAR AVE, SUITE 2904
MEMPHIS TN 38137

********** PRODUCT NAME **********

**03080* CALCIUM ARSENATE

**07878* CALCIUM ARSENATE

**** PRODUCT SEARCH LISTING **** PAGE 3

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 005967 MOYER CHEMICAL COMPANY
BOX 945
SAN JOSE CA 95108

********** PRODUCT NAME **********

**05152* SODIUM ARSENITE NO. 4-L

****

REGISTRANT *NAME AND ADDRESS*

* 010193 SHEPP CHEMICAL & SUPPLY COMPANY
2827 RIVERVIEW BLVD
BRADENTON FL 33505

********** PRODUCT NAME **********

**09447* NO-GRO LIQUID CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 010827 INDUSTRIAL SOLVENTS
PO BOX 312
SAN MARCOS TX 78666

********** PRODUCT NAME **********

**04073* IND-SOL 40

**** PRODUCT SEARCH LISTING **** PAGE 4

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 011134 PEST CONTROL CHEMICALS INC
5852 S WESTERN AVE
LOS ANGELES CA 90047

********** PRODUCT NAME **********

**06022* PESTCO K-A-L TERM DUST

****

REGISTRANT *NAME AND ADDRESS*

* 011151 ROBINSON LAB INC
355 BRANNAN ST
SAN FRANCISCO CA 94107

********** PRODUCT NAME **********

**03820* MATTES ANT PASTE

****

REGISTRANT *NAME AND ADDRESS*

* 013437 DU COR CHEMICAL CORP.
P.O. BOX 13298
ORLANDO, FL 32809

********** PRODUCT NAME **********

**04074* PERMAKILL'WEED KILLER CONCENTRATE

**04075* DU COR FORMULA 71 WEED KILLER CONCENTRATE

**** PRODUCT SEARCH LISTING **** PAGE 5

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 018599 BARKER LABORATORIES
1200 S BROAD AVE
NEW ORLEANS LA 70125

********** PRODUCT NAME **********

**10168* KILROY WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 035053 HONOLULU WOOD TREATING CO
2290 ALAHAE PLACE
HONOLULU, HI 96819

********** PRODUCT NAME **********

**05213* WOLMAN WOOD PRESERVATIVE SOLUTION

**05242* WOLMANAC II SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 036019 OSMOSE PACIFIC, INC
2819 PUKOLOA STREET
HONOLULU, HI 96819

********** PRODUCT NAME **********

**03490* OSMOSE SPECIAL K-33 PRESERVATIVE

**** PRODUCT SEARCH LISTING **** PAGE 6

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT *NAME AND ADDRESS*

* 036686 BIG BEE CHEMICAL AND SUPPLY CO.
ROUTE 4 OLD CONYERS ROAD
STOCKBRIDGE, GA 30281

********** PRODUCT NAME **********

**08218* 40% SODIUM ARSENITE SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 037347 UNI-CHEM CORP. OF FLORIDA
BOX 6336 - 2801 N.W. 55TH CT.
FT. LAUDERDALE, FL 33310

********** PRODUCT NAME **********

**C4050* WEED-X

****

REGISTRANT *NAME AND ADDRESS*

* 037843 UNIVERSITY OF HAWAII-CO-OP
EXTENSION SERV-ENTOMOLOGY BRANCH
HONOLULU, HI 96822

********** PRODUCT NAME **********

**08562* PARIS GREEN

**** PRODUCT SEARCH LISTING **** PAGE 7

08/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING INORG. ARSENICALS

****

REGISTRANT	*NAME AND ADDRESS*
* 038524	RICHARD LUDWIG 6720 EAST VALLEY VISTA LANE PARADISE VALLEY, AZ 85253

********** PRODUCT NAME **********

**10224* K-9 ANT POISON

[FR Doc. 78-28935 Filed 10-17-78; 8:45 am]

[6560-01-M]

[FRL 984-2; OPP-30000/30]

PESTICIDE PROGRAMS

Notice of Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing Pentachlorophenol

AGENCY: Office of Pesticide Programs, Environmental Protection Agency (EPA).

ACTION: Notice of rebuttable presumption.

SUMMARY: Pentachlorophenol and its derivatives (henceforth referred to as PCP) have been found to exceed certain risk criteria set forth in 40 CFR 162.11. This notice requests registrants and other interested persons to submit rebuttals and other information on the presumption and to submit any other data on the risk and benefits of this pesticide chemical. This notice is the first of several which will give public notification of the Agency's progress in reviewing this chemical. Today EPA is also publishing in the FEDERAL REGISTER notices of rebuttable presumption against registration (RPAR) on Inorganic arsenic and coal tar, creosote, and coal tar neutral oil. inorganic arsenic, PCP, and coal tar and derivatives comprise the most widely used of pesticide wood treatments available. Therfore, the wood treatment uses of these pesticides will be considered together in the risk/benefit part of the RPAR review process. Other uses for these pesticides will be considered in individual risk/benefit analyses for each chemical.

DATE: Rebuttal evidence and other information must be received on or before December 4, 1978.

ADDRESS MATERIAL TO: Federal Register Section, Program Support Division (TS-757), Office of Pesticide Programs, EPA, Room 401, East Tower, 401 M Street SW., Washington, D.C. 20460.

FOR FURTHER INFORMATION CONTACT:

Daniel P. Cirelli, Special Pesticide Review Division, Office of Pesticide Programs (TS-791), Room 447, East Tower, EPA, 202-755-2972.

SUPPLEMENTARY INFORMATION: The Deputy Assistant Administrator, Office of Pesticide Programs, EPA, has determined that a rebuttable presumption exists against registration and continued registration of all pesticide products containing pentachlorophenol.[1]

Issuance of this RPAR means that potential adverse effects associated with the use of pentachlorophenol have been identified and will be examined further to determine if they do exist and, if so, whether they are unreasonable. This process involves a public review of both the risks and the benefits associated with the uses of the pesticide. The RPAR does not mean that the Agency has decided to cancel or to take any other action with respect to it; EPA has not made a finding that use of any products containing this ingredient presents an imminent hazard. Any pesticide subject to this RPAR may lawfully be sold, distributed, and used for all uses for which it is registered and labeled.

I. REGULATORY PROVISIONS

A. *General.* Title 40, Part 162.11, of the Code of Federal Regulations for the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) as amended (86 Stat. 973, 89 Stat. 751, 7 U.S.C. 136 et seq.), provides that a rebuttable presumption against registration shall arise if the Agency determines that a pesticide meets or exceeds any of the risk criteria relating to acute and chronic toxic effects set forth in section 162.11(a)(3). If it is determined that such a rebuttable presumption has arisen, the regulations require that the registrant be notified by certified mail and afforded an opportunity to submit evidence in rebuttal of the presumption. In addition, the Agency has determined that the public should also be given notice of the bases for the presumption to provide an opportunity for comment and to solicit additional information relevant to the presumption.

A notice of rebuttable presumption against registration is issued when the evidence related to risk meets the criteria set forth in section 162.11(a)(3). It is emphasized that a notice of rebuttable presumption against registration and continued registration of a pesticide is not a notice of intent to cancel the registration of a pesticide, and may or may not lead to cancellation. The notice of intent to cancel is issued only after the risks and benefits of a pesticide are carefully considered and it is determined that the pesticide may generally cause unreasonable adverse effects to the environment.

All registrants and applicants for registration are invited pursuant to 40 CFR 162.11(a)(4) to submit evidence in rebuttal of the presumptions listed in part II of this notice and, in the case of oncogenicity, to submit information which relates to the assessment of oncogenic risks as set forth in the Agency's Interim Procedures and Guidelines for Health Risk and Economic Impact Assessment of Suspected Carcinogens (May 25, 1976; 41 FR 21402). Registrants and other interested parties may submit for consideration data on benefits which they believe would justify registration or continued registration. In addition, any registrant may petition the Agency to voluntarily cancel a current registration pursuant to section 6(a)(1) of FIFRA.

B. *Rebuttal criteria.* Section 162.11(a)(4) provides that a registrant may rebut the presumption by sustaining the burden of proving:

(1) In the case of a pesticide presumed against pursuant to the acute toxicity or lack of emergency treatment criteria, "that when considered with the formulation, packaging, method of use, and proposed restrictions on and directions for use and widespread and commonly recognized practices of use, the anticipated exposure to an applicator or user and to local, regional, or national populations of nontarget organisms is not likely to result in any significant acute adverse effects" (40 CFR 162.11(a)(4)(i));

(2) In the case of a pesticide presumed against pursuant to the chronic toxicity criteria, "that when considered with proposed restrictions on use and widespread and commonly recognized practices of use, the pesticide will not concentrate, persist or accrue to levels in man or the environment likely to result in any significant chronic adverse effects" [40 CFR 162.11(a)(4)(ii)]; or

(3) In either case, that "the determination by the Agency that the pesticide meets or exceeds any of the criteria for risk was in error" [40 CFR 162.11(a)(4)(iii)].

C. *Benefits information.* In addition to submitting evidence to rebut the presumption of risk, section 162.11(a)(5)(iii) provides that a registrant "may submit evidence as to whether the economic, social and environmental benefits of the use of the pesticide subject to the presumption outweigh the risk of use." If the risk presumptions are not rebutted, the benefit evidence[2] submitted by the

[1] A position document, containing an appendix of references, background information, and other material pertinent to the issuance of this notice, has been prepared and is also published with this notice.

[2] Registrants or other interested persons who desire to submit benefit information should consider submitting information on the following subjects, along with any other relevant information they desire to submit:

1. Identification of the major uses of the pesticide, including estimated quantities used by crop or other application.
2. Identification of the minor uses of the pesticide, including estimated quantities used by category such as lawn and garden uses and household uses.
3. Identification of registered alternative products for the uses set forth in (1) and (2) above, including an estimate of their availability.
4. Determination of the change in costs to the user of providing equivalent pesticide treatment with any available substitute products.

Footnotes continued on next page

registrant, applicants, and other interested persons will be considered by the Administrator in determining the appropriate regulatory action. Specifically, section 162.11(a)(5)(iii) provides that if the "benefits appear to outweigh the risks," the Administrator may issue a notice of intent to hold a hearing pursuant to section 6(b)(2) of FIFRA to determine whether the registration(s) should be canceled or application(s) denied. Alternatively, if the "benefits do not appear to outweigh the risks, the Adminstrator shall issue a notice pursuant to section 3(c)(6) or section 6(b)(1) of the Act, as appropriate." Moreover, if at any time the Administrator determines that a pesticide poses an "imminent hazard" to humans or the environment, a notice of suspension may be issued pursuant to section 6(c) of the Act.

II. Presumptions

Registrations and applications for registration of pesticide products containing pentachlorophenol meet or exceed the 40 CFR 162.11(a)(3) risk criteria relating to teratogenic and/or fetotoxic effects in mammalian test species. The Agency's basis for concluding that these risk criteria have been met or exceeded is set out in "Pentachlorophenol: Position Document 1," which follows. Copies of attachments to the position document which are not published with this notice are available for public inspection in the office of the Special Pesticide Review Division. Information protected from disclosure pursuant to FIFRA Section 10 cannot be provided. Specific inquiries concerning the position document, as well as requests for access to these files, should be directed to Project Manager Daniel Cirelli, Special Pesticide Review Division (TS-791), EPA, Room 447, East Tower, 401 M Street SW., Washington, D.C. 20460, 202-755-2972.

A. *Chronic or delayed toxic effects.* 40 CFR Section 162.11(a)(3)(ii)(B) provides that a rebuttable presumption shall arise if a pesticide "(p)roduces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety * * *."

On the basis of scientific studies and information summarized in the position document, the Agency has concluded that registrations and applications for registration of pesticide products containing pentachlorophenol exceed this risk criterion for teratogenicity and fetotoxicity and that a rebuttable presumption against new or continued registration of such products has arisen.

III. Additional Grounds for Review

As discussed in detail in the attached position document, some data has associated pentachlorophenol with mutagenic effects in test organisms. The data and analyses available at this time with respect to this effect are not sufficient to warrant the issuance of a rebuttable presumption. The Agency specifically solicits further evidence bearing on these possible adverse effects. All comments and information received with respect to the potential adverse effects, including analysis thereof, may serve as a basis for a final decision on registering pesticide containing pentachlorophenol.

IV. Rebuttal Submission Procedures

All registrants and applicants for registration listed below are being notified by certified mail of the rebuttable presumption existing against registration and continued registration of their products.

The registrants and applicants for registration shall have 45 days from the date this notice is sent or until December 4, 1978, to submit evidence in rebuttal of the presumption. However, the Administrator may, for good cause shown, grant an additional 60 days during which such evidence may be submitted. Notice of such an extension, if granted, will appear in the Federal Register.

A registrant or applicant for registration may, if it desires, assert a business confidentiality claim covering part of all of the information submitted in rebuttal. The registrant or applicant may assert the claim by placing on or attaching to the information a cover sheet, stamped or typed legend, or other suitable form of notice employing language such as "trade secret," "proprietary," or "company confidential." Allegedly confidential protions of otherwise nonconfidential documents should be clearly marked.

If a confidentiality claim is asserted, the information covered by the claim will be disclosed by EPA only to the extent and by means of the procedures set forth in 40 CFR Part 2, Subpart B (41 FR 3690; September 1, 1976). If no confidentiality claim accompanies the information at the time it is received by EPA, EPA will place the information in the public comment file where it will be available for public inspection.

If a registrant or applicant does assert a confidentiality claim for some but not all, of the information submitted to EPA in rebuttal, the registrant or applicant should furnish two copies of the information to EPA. The first copy should contain all of the information submitted in rebuttal with information claimed as confidential clearly identified. The second copy should be identical to the first except that all information claimed as confidential should be deleted. The second copy will be placed in the public comment file. The first copy will be treated in accordance with the procedures set out above.

V. Duty To Submit Information on Adverse Effects

Registrants are required by law to submit to EPA any additional information regarding any adverse effects on man or the environment which comes to a registrant's attention at any time, pursuant to section 6(a)(2) of FIRFRA and 40 CFR 162.8(d). If any registrant of pentacholorophenol products has any published or unpublished information, studies, reports, analyses, or reanalyses regarding any adverse effects in animal species or humans, residues, and claimed or verifed accidents to humans, domestic animals, or wildlife, which have not been previously submitted to EPA, the material must be submitted immediately. When responding to this notice, each registrant shall submit a written certification to the Agency that all information regarding any adverse effects known to the registrant has been submitted. In addition, the registrants should notify EPA of any studies currently in progress, including the purpose of the study, the protocol, the approximate completion date, and a summary of all results observed to date.

VI. Public Comments and Inspection

During the time allowed for submission of rebuttal evidence, specific comments on the presumptions set forth in this notice and on the material contained in the position document are solicited from the public. In particular, any documented episodes of adverse effects to humans, domestic animals, or wildlife, and information as to any laboratory studies in progress or completed are requested to be submitted to EPA as soon as possible. Specifically, information on the fate and effects of pentachlorophenol, its impurities, metabolites, and degradation products on flora and fauna, particularly animals with metabolism similar to man, is solicited. Similarly, any studies or comments on the benefits from the use of pentachlorophenol are requested to be submitted. All comments and information received, as well as any other relevant information

---

Footnotes continued from last page

5. Assessment of regulation impact upon user productivity (e.g., yield per acre and/or total output) from using available substitute pesticides or from using no other pesticides.

6. If the impacts upon either user costs or productivity are significant, a qualitative assessment of the regulation's impact on production of major agricultural commodities and retail food prices of such commodities.

and analysis thereof, which come to the attention of the Agency may serve as a basis for final determination pursuant to § 162.11(a)(5).

All comments and information should be sent to the office of the Federal Register Section at the address given above, it possible in triplicate to facilitate the work of the Agency and others interested in inspecting them. The comments and information should bear the identifying notation "OPP-30000/30." Comments received after the specified time period will be considered only to the extent feasible, consistent with the time limits imposed by 40 CFR 162.11(a)(5)(ii).

All written comments and information filed pursuant to this notice will be available for public inspection in the office of the Federal Register Section from 8:30 a.m. to 4 p.m. during normal working days. Interested persons are encouraged to take advantage of the opportunity to inspect Agency files during normal working hours since (1) all of the information received may serve as a basis for final determination pursuant to § 162.11(a)(5) and (2) the Agency will not generally publish a summary of information received in the FEDERAL REGISTER at the close of the rebuttal period.

Your cooperation is solicited in identifying any errors or omissions which may have been made in the following computer listings. Corrections to the listings may not necessarily be published in the FEDERAL REGISTER, but rather handled by mail with affected parties. Omissions will be corrected by notice in the FEDERAL REGISTER.

Dated: September 30, 1978.

EDWIN L. JOHNSON,
Deputy Assistant Administrator for Pesticide Programs.

[6560-01-C]

PENTACHLOROPHENOL

POSITION DOCUMENT 1

Daniel P. Cirelli, Project Manager
U.S. Environmental Protection Agency

I. Background

A. Chemistry

1. General

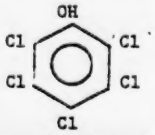
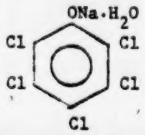
Pentachlorophenol (PCP or PENTA)[1/] is a buff-colored crystal which is produced in the United States by chlorination of molten phenol in the presence of a catalyst. Derivatives of PCP which are registered for use as pesticides are the sodium and potassium salts (Na-PCP and K-PCP, respectively), and the lauric acid ester (L-PCP). The structures and physical properties of PCP and Na-PCP are given in Figure I. K-PCP is produced as in situ formulations by dissolving PCP in potassium hydroxide solutions. The physical and chemical properties of pure K-PCP are not available from standard chemical references or from EPA files. L-PCP is produced by esterification of PCP with mixtures of alkyl carboxylic acids, the most predominant of which is lauric acid. Technically pure L-PCP is a brown oil having a specific gravity of 1.28 at room temperature. It is soluble in non-polar solvents, oils, fats, waxes and plasticizers, and insoluble in water and alcohols.

Industrial production of PCP is a two-stage process. In the first stage, isomers of tri- and tetrachlorophenols are formed when the reaction temperature is about 105 °C. In the second stage, the temperature is progressively increased to 130 °C to keep the reaction mixture molten, and the tri- and tetrachlorophenols are further chlorinated to form PCP. This reaction is not quantitative; tetrachlorophenols persist during the reaction and are carried with PCP during subsequent processing. The result is that

1/ PCP will be used as an abbreviation for pentachlorophenol in this document.

Figure I

Physical properties of pentachlorophenol and sodium pentachlorophenate

Structural Formula		
Name	Pentachlorophenol	Sodium Pentachlorophenate
Formula	C_6Cl_5OH	$C_6Cl_5ONa \cdot H_2O$
Molecular Weight	266.4	306.3
Description	Buff-colored crystal	Buff-colored crystal
Specific Gravity	1.9	2.0
Density	1.987	-
Vapor Pressure	0.00015 (25 °C)	-
Solubility, g/100 g, 25 °C		
Water	< 0.01	33
Acetone	50	35
Benzene	15	-
Diaetone Alcohol	190	45
Ethanol (95%)	120	65
Methanol	180	25
Isopropanol	85	30
Ethylene glycol	11	40

technical grade PCP contains from 4 to 12% tetrachlorophenols. These tetrachlorophenols are listed on the product labels as active ingredients. One of the three possible tetrachlorophenol isomers, 2,3,4,6-tetrachlorophenol, was registered by the Dow Chemical Company as Dowicide 6, a fungicide; this chemical is no longer produced as a separate product. It is, however, listed as an active ingredient in some PCP products.

The elevated temperatures required for the second stage of PCP production favor condensation of the tri- and tetrachlorophenols to form hexa-, hepta-, and octa-chlorodibenzo-p-dioxins (dioxins) and various chlorinated dibenzofurans (furans). These impurities are also carried forward with PCP and will be discussed in greater detail in Section I.C.

2. Environmental Residues

The amount of PCP produced in the United States, approximately 50,000,000 pounds/year, and its widespread use as a pesticide strongly indicate its potential as an environmental pollutant. Possible sources of PCP in the environment are: runoff from manufacturing or wood preserving processes, leaching or vaporization from preserved wood, and from its use as a termiticide and herbicide. Although there is evidence that PCP could be found in the environment, the ambient monitoring programs for air, water, and soils do not routinely test for PCP.

a. Air

The vapor pressure of PCP, 0.00017 torr at 20 °C and 0.0031 torr

at 50 °C, suggests that it can volatilize from treated surfaces (torr: pressure required to support 1 mm of mercury at 0 °C). Gebefugl et al. (1976) measured PCP vapors in a closed test area in which wall paneling was covered according to label instructions with a wood-protecting agent containing PCP. Air samples were taken every day for 9 days at 25 °C, and then for 6 days at 28 °C. Samples were taken in the morning, and the sampling period was not specified. Gas chromatographic measurements showed PCP measurements ranging from 1 microgram/cubic meter (ug/m^3) on the first day to 160 ug/m^3 on the fourth day. There were wide fluctuations in PCP concentrations during the course of the experiment. In a separate experiment, an increase in the amount of water vapor in the test area decreased the PCP levels in the air.

Bevenue et al. (1972) measured PCP in rain water in Hawaii [2-284 parts per trillion (ppt)], in snow samples taken from Mauna Kai Summit (14 ppt), and in lake water fed by this snow (10 ppt). The authors did not determine whether this airborne PCP was in gaseous form or adsorbed on dust particles. Because of the heavy use of PCP in Hawaii to protect wooden surfaces against termites, both physical forms are likely to occur in the air.

There is indirect evidence that PCP vapors can be toxic. An article that appeared in California Health in 1970 (Anon. "Pentachlorophenol Poisoning in the Home") described the effects on the inhabitants of painting interior wood paneling with PCP-containing paint. House plants within the

home died in 3 or 4 days. Over a 3-month period, the housewife became progressively weaker while losing 20 pounds. She recovered after hospitalization. Her condition was diagnosed as PCP poisoning by a California State Department toxicologist.

Ferguson in 1959 showed that PCP vapors were toxic to conifer seedlings grown in greenhouses on wood flats whose sideboards had been treated with PCP. Leaching of PCP into the soils of the flats was not implicated since transfer of these soils to untreated flats supported normal seedling growth. He observed toxic effects, to a lesser extent, on seedlings in untreated flats, which he attributed to air movement from treated to the untreated flats. PCP has been found in the blood and urine of workers in wood-preserving factories; in one report this was attributed, in part, to respiratory exposure (Casarett et al., 1969).

The American Conference of Government Industrial Hygienists has established a PCP Threshold Limit Value-Time Weighted Average of 0.5 mg/m^3 for a normal 8-hour workday or 40-hour workweek, and a Threshold Limit Value-Short Term Exposure Limit of 1.5 mg/m^3 for a 15-minute period.

b. Water

PCP can be released into water as an effluent from manufacturing or wood-preserving plants, by leaching from treated wood exposed to rain, and by runoff from its uses as a herbicide, fungicide, and molluscicide. In spite of its wide production and variety of uses, PCP is not routinely monitored by the National Water Monitoring Program, which measures contaminants in rivers and streams in the United States. This program reported fifteen measurements of PCP along the California Aqueduct between 1974 and 1976. There was no sampling previous to 1974. Values reported from five sites along the Aqueduct ranged from 0.01 to 16.0 ug/liter PCP in whole water samples.

Buhler et al. (1973) measured PCP in sewage treatment plant effluent collected simultaneously from three Oregon cities. He reported PCP levels between 1 and 4 parts per billion (ppb). The same workers found PCP concentrations between 0.10 and 0.70 ppb in water sampled from the Willamette River, and 0.6 ppb in drinking water from a water treatment plant. In unpublished data, the EPA Drinking Water Program has found PCP in the water of 86 of the 108 cities sampled; the mean concentration of the positives was 0.07 ug/liter. The median concentration for all cities was 0.051 ug/liter (Kutz, 1977).

In 1976 Fountaine et al. analysed samples from a stream which originated in an area containing several manufacturing plants, including a wood-preserving installation. The highest measurement of PCP, 5,450 parts per million (ppm), was in surface oil slicks; water samples ranged from 0.082 to 10.5 ppm. The authors noted that these PCP levels are not necessarily due to discharge from the wood-preserving factory outlet into the stream. The factory had been in business for 20 years. Gross spillage of PCP occurred in the factory area, particularly in the early years of operation, and this PCP seeped into the ground. There is, therefore, the possibility that the PCP levels in the stream were due to runoff from PCP-contaminated soil, rather than direct discharge from

the factory. Measurements of PCP in the factory discharge were not reported.

c. Soils

Because the National Soils Monitoring Program does not routinely analyze soil samples for PCP, ambient levels in soils are not available.

In a series of papers, Choi and Aomine (1972, 1974a, 1974b) studied the adsorption properties of PCP on various soil types and under a variety of environmental conditions in the laboratory. They found that PCP was adsorbed to some extent on all soil types. Soil acidity was a major factor in determining the amount of PCP adsorbed; strongly acidic soils (pH 5) adsorbed more than less acid soils (pH 6). There was less adsorption by weakly acidic or neutral soils. However, increased organic matter or temperature or both increased PCP adsorption at the pH-values tested. In a series of experiments using different soil types and ion concentrations, the authors demonstrated competition between inorganic ions and PCP for adsorption sites on soil colloids. They concluded from their experiments that PCP is adsorbed on soil through ion exchange reactions as well as molecular adsorption due to van der Waals forces.

The fate of PCP in soils has been extensively studied. The primary factor in the degradation of PCP in soils is microbiological activity; other contributing factors include soil type, moisture content, and temperature. Kuwatsuka and Igarashi (1975) demonstrated a positive correlation between the proportion of organic matter and the degradation rate of PCP in soils, observing that soils devoid of organic matter did not degrade PCP. Ide et al. (1972) found that PCP did not degrade in sterilized soil samples; Young and Carroll (1951) showed that PCP decays most rapidly in soil containing a large proportion of organic matter and at temperatures which are optimal for microbiological activity. Kuwatsuka and Igarashi (1975) compared the PCP degrading characteristics of upland (i.e., aerated) soil with flooded (i.e., rice paddy) soil. They found that soil microorganisms degraded PCP most rapidly when they were maintained in the environment to which they are adapted, e.g. flooding upland soil and aerating flooded soil decreased the rate of PCP decay. Using a gas chromatograph with an electron-capture detector the authors identified tri- and tetrachlorophenols as degradation products of PCP and indicated that, at least as a first step, PCP degrades by dechlorination. Ide et al. (1972) found tri- and tetrachloroanisoles as PCP degradation products. This indicated that methylation is also involved in the degradation process.

The persistence of PCP in soils ranges between 21 days and 5 years, depending primarily on the microbial population. Watanebe (1973) reported complete degradation in paddy soil in 21 days. Hetrick (1952) claimed that PCP persisted for over 5 years in an unspecified soil type.

d. Plants and Animals

i. Plants

There are no data available on PCP residues in higher plants resulting from its use as a pesticide.

ii. Animals

In 1974 Vermeer et al. reported the death of wildlife caused by application of Na-PCP to a rice field in Surinam, South America. The Na-PCP was applied at the rate of 4 kg/hectare to control water snails which damaged young rice plants. PCP residues were measured in snails (36.8 mean wet-weight ppm), frogs (8.1 mean wet-weight ppm), fishes (31.2, 41.6 and 59.4 mean wet-weight ppm for three species), and snail kites, a bird which feeds on water snails. The dead kites showed mean PCP residues of 11.25 ± 1.11 ppm in brain tissue, 45.56 ± 2.18 ppm in liver, and 20.34 ± 1.25 ppm in the kidneys, all on a wet weight of tissue basis. These PCP levels were 53, 74, and 166 times greater, respectively, than those in kites collected from an untreated marsh. The investigators attributed the death of the kites from the treated field to their feeding on water snails containing PCP. In addition, when other bird species which frequented the treated fields were examined, PCP was detected at levels ranging from 0.04 to 0.24 wet-weight ppm in the liver and 0.08 to 0.49 wet-weight ppm in the brain.

Early in 1977 PCP was detected in the blood of dairy cattle and calves on eight Michigan farms. These herds all showed signs of sickness and had high calf mortality rates. The levels of PCP found in one herd ranged from 270 to 570 ppb. This herd was housed in a total-confinement barn which had been constructed, in part, of PCP-treated wood. In another herd the dioxins found in PCP were detected in fat and liver tissue in the parts per billion and parts per trillion ranges, respectively. As a result of this incident, the Animal and Plant Health Inspection Service of the Department of Agriculture (USDA) instituted a nationwide survey of beef fat and liver for the presence of the hexa- and octachlorodioxins found in PCP. In the first group of 238 beef samples collected in seventeen States, 70 (29.4%) showed positive levels using low resolution mass spectrometry. Of these 70 samples, 4 had been confirmed using high resolution mass spectrometry. Detection levels were in fractional nanograms/gram for both hexa- and octa-chlorodibenzo-*p*-dioxins. The determination of the signficance of these residues awaits the completion of the survey and its subsequent statistical and methodological analysis.

e. Humans

PCP appears to be ubiquitous in human urine. In 1970 Cranmer and Freal found PCP in urine at concentrations ranging from 2 to 11 ppb in the general population. In 1967 Bevenue et al. found PCP in the urine of 130 Hawaiians occupationally exposed to PCP at levels ranging from 0.03 to 35.7 mg/liter. In the same study, PCP was found in the urine of all but one of 117 unexposed subjects; these values varied from 0 to 0.44 mg/liter. In a recent study, preliminary data from a joint U.S. Public Health Service - EPA effort disclosed that 86% of the urine samples taken from subjects within the continental United States showed positive PCP values averaging 6.3 ppb, with a maximum observed value of 193 ppb. The limit of detection was 5 ppb (Kutz et al., 1978).

Arsenault (1976) reported the mean blood serum level of PCP in twenty-one workers in pressure treatment plants to be 1.05 ppm; there

were about 0.1 ppm in the controls (unspecified). Data for the general population are not available. In another study Shafick (1973) reported PCP levels ranging from 12 to 52 ppb in the adipose tissue of eighteen subjects who were not occupationally exposed to PCP. Dougherty and Piotrowska (1976) detected PCP in seminal fluid from seven sexually active men. Levels detected averaged 50 ppb (range: 20-70 ppb).

The sources and exposure routes of PCP residues in human tissues have not been conclusively established. Possible routes are inhalation of vaporized PCP and ingestion of PCP in water and food. In addition, PCP forms as a metabolic product of hexachlorobenzene (HCB). Lui and Sweeney (1975) fed rats 0.25% HCB, which amounted to about 50 to 100 mg HCB daily. Analysis of urine by gas-liquid chromatography showed a level of 12.5 ug/ml PCP in a daily urine output of about 15 ml. The analyses were confirmed by gas-liquid chromatography/mass spectrometry.

The Food and Drug Administration Market Basket Survey has estimated the amount of pesticides in foodstuffs since 1965. The estimates are based on the average daily intake of an 18-year-old male, whose food consumption is the highest of any age group of either sex. The most recent data from this survey (Johnson and Manske, 1977) are available from food samples collected from August 1974 to July 1975. PCP was assayed in twelve food commodities collected in twenty cities in the United States, so that there were twenty measurements for each commodity. The results are summarized in Table 1.

TABLE 1. Pentachlorophenol in food commodities 1/

Commodity	Average, ppm 2/	Positive Composites	Range, ppm
Dairy Products	0.0005	1	0.01
Meat, Fish, and Poultry	-	0	-
Grain and Cereal Products	0.001	2	0.01-0.013
Potatoes	-	0	-
Leafy Vegetables	T 3/	1	T
Legume Vegetables	-	0	-
Root Vegetables	0.001	2	0.010
Garden Fruits	T	1	T
Fruits	T	1	0.011
Oils, Fats, and Shortening	-	0	-
Sugars and Adjuncts	0.006	5	0.01-0.04
Beverages	-	0	-

1/ Data from Johnson and Manske (1977).
2/ Averages based on twenty composite samples; trace residues were not included in calculating the average.
3/ T = trace.

These low levels of PCP in food are a constant means of exposure to humans. However, the connection between this route of exposure and the presence of PCP in blood, urine, and adipose tissue is not clear.

B. Metabolism

1. Plants

As described earlier, microorganisms are capable of degrading PCP in soils. Certain bacterial strains have been isolated which are capable of growing on media containing PCP (Morton et al., 1969) and of using PCP as the sole source of carbon (Chu and Kirsch, 1972). The metabolic mechanisms of bacterial action on PCP are, however, unknown. Certain species of fungi, some of which are present in wood, have been found to tolerate PCP. Several of these fungi depleted PCP in treated wood blocks, but lost their ability to cause wood decay (Duncan and Deverall, 1964). As is the case with bacteria, metabolic pathways of PCP degradation by fungi have not been identified. There are no data available on the metabolism of PCP by higher plants.

2. Animals

There have been two studies on PCP metabolism in aquatic animals. Each of these papers demonstrated the same metabolic pathway. In 1970 Kobayashi et al. reported that the short-necked clam (Tapes phillippinarium) converts PCP to pentachlorophenyl sulfate in sea water. The level of PCP in the shellfish reached a plateau after about 24 hours of exposure. After 50 hours, 80% of the PCP had been converted to the sulfate conjugate. The same detoxification mechanism was demonstrated by Akitake and Kobayashi

(1975) in goldfish cultured in fresh water containing PCP. Whether this detoxification mechanism is common to other aquatic animals is not known.

Mammals are known to excrete PCP in unchanged form after exposure by various routes. In 1974 Ahlborg et al. administered ^{14}C-PCP orally and intraperitoneally to rats and mice (10 to 25 mg/kg). Approximately 40% was excreted as PCP in the urine. ^{14}C-tetrachlorohydroquinone was also detected at levels equal to 5% of the excreted radioactivity in rats, and 24% of the activity in mice. The authors also found PCP and tetrachlorohydroquinone in the urine of workers occupationally exposed to PCP. Larsen et al. (1972) orally administered ^{14}C-PCP to rats, and found that 50% of the radioactivity was excreted in the urine in 24 hours, 68% was excreted in 10 days; between 9.2 and 13.2% was excreted in the feces. Tissue analyses 40 hours after exposure showed small amounts of ^{14}C activity in the adrenal glands, blood, brain, fat, heart, kidneys, liver, lungs, muscles, ovaries, spleen, stomach and intestine, and testes. Highest levels were found in liver (0.23% of administered radioactivity), kidney (0.18%), and blood (0.15%). In blood, 99% of the radioactivity detected was in the serum.

Larsen et al. (1972) postulated a two-component urinary excretion pattern that has a 10-hour half-life for the first 2 days, followed by a 102-day half-life. In 1969 Casarett et al. measured PCP concentrations in the urine of two subjects after respiratory exposure and determined that after 24 hours, the half-life is approximately 10 hours. They found a blood-to-urine PCP ratio of between 1.5 and 2.5 in occupationally ex-

posed individuals. At about 10 ppm, PCP in blood plasma reached a plateau while urine levels continued to increase. They postulated that PCP binds to plasma protein, and is subsequently distributed to the tissues. The work of Larsen et al. (1972) seems to confirm this.

Tashiro et al. (1970) demonstrated an additional metabolic pathway for PCP. They collected and analysed the urine of three rabbits fed 10 g of unlabeled PCP over 30 days. They identified a sugar conjugate of PCP, pentachlorophenyl-beta-glucuronide, in the urine which led them to propose a three-way excretory pathway for PCP in mammals: unchanged PCP; the oxidation product "chloranil", which is actually tetrachloroquinone, and the conjugate PCP-beta-glucuronide.

C. Pentachlorophenol Contaminants

As stated previously, commercial production of PCP results in the formation of dioxin and furan contaminants. Figure 2 shows the structural formula of the basic molecules. Substitution of chlorine atoms at one or more of the numbered positions results in the chlorinated dibenzo-p-dioxin (dioxin) and chlorinated dibenzofuran (furan) chemical families. There are 75 members of the dioxin family and 135 furans. Members of these families differ not only in the number of chlorine atoms on the molecule, but also in the positioning of these atoms (isomerism). Thus the dioxin that has six chlorine atoms on its molecule has ten possible isomers.

The dioxins which are found in PCP are the hexa- (HCDD), hepta- (HeCDD), and octa-chlorodibenzo-p-dioxins (OCDD); the furans found are

FIGURE 2. Structure of dioxin and furan

the tetra-, penta-, hexa-, hepta-, and octa-dibenzofurans (Buser and Bosshardt, 1976). The amount of these chemicals in PCP varies with each industrial batch produced, even when produced by the same manufacturer. Analytical methods for measuring individual isomers require sophisticated and expensive analytical instrumentation, and standards for the isomers are not generally available. Until recently, both the dioxins and furans in PCP have been measured as isomeric groups. Table 2 shows the composition of typical commercial PCP.

The physical, chemical, and toxicological properties of individual dioxins and furans are, with one exception, relatively unknown. The exception is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), which has been shown to be one of the most toxic compounds known. TCDD is not found in PCP at a limit of detection less than 0.05 ppm (Johnson et al., 1973). However, the dioxins in PCP have been shown by Johnson et al.

TABLE 2. Composition of commercial pentachlorophenol 1/

Component	Dowicide 7	Dowicide EC-7 2/
Pentachlorophenol	88.4%	89.8%
Tetrachlorophenol	4.4%	10.2%
Trichlorophenol	< 0.1%	< 0.1%
Chlorinated Phenoxyphenols	< 6.2%	-
Hexa-dioxins	4 ppm	1.0 ppm
Hepta-dioxins	125 ppm	6.5 ppm
Octa-dioxin	2500 ppm	15.0 ppm
Hexa-furans	30 ppm	< 1 ppm
Hepta-furans	80 ppm	1.8 ppm
Octa-furan	80 ppm	< 1 ppm

1/ Data from Dow Chemical Company.
2/ Total greater than 100% because numbers are rounded off.

(1973) to cause chloracne in rabbits and edema in chicks. The only data available comparing the toxic effects of individual dioxin isomers were reported by McConnel et al. (1977), and these are shown in Table 3. These data indicate that the hexa- and hepta-dioxin isomers tested were less toxic than TCDD in mice and guinea pigs; however, they would be classified as Category I poisons (oral LD-50 less than 50 mg/kg).

TABLE 3. Single oral LD-50-30 of dioxin isomes 1/

Isomer	LD-50-30, ug/kg Guinea Pigs	Mice
1,2,3,4,7,8-HCDD	72.5	825
1,2,3,6,7,8-HCDD	70-100 2/	1250
1,2,3,7,8,9-HCDD	60-100 2/	1440
1,2,3,4,6,7,8-HeCDD	7180	-
TCDD 3/	2	283.7

1/ Data from McConnell et al. (1977).
2/ Estimated range represents variability among replicates.
3/ TCDD values are shown for comparative purposes.

Firestone (1977) detected PCP, individual hexa- and hepta-dioxin isomers, and octa-dioxin in samples of commercial gelatin procurred from supermarkets and in bulk. Gelatin is produced from pork skins and cattle

bones and hides, which may be preserved with PCP during processing both in this country and abroad. According to Firestone, the annual consumption of gelatin in the United States is 57 million pounds of domestic production and 13 million pounds of imports.

Highest levels of PCP and total dioxins were found in bulk gelatin that was imported from Mexico and produced by a single company. Three measurements of this gelatin averaged 6.4 ppm of PCP, and six measurements averaged 26.8 ppb of total dioxins. In contrast, bulk domestic porkskin gelatin had no PCP and 0.1 ppb of total dioxins (octadioxins) in one of two sample measurements. Three consumer packages of unflavored gelatin purchased in a supermarket showed 0.2, 0.8, and 3.6 ppb of total dioxins.

D. Registered Uses and Production

Pentachlorophenol and its derivatives are among the most versatile pesticides now in use in the United States. This versatility is due first to their efficacy against a wide variety of pests (bacteria, yeast, slime molds, algae, fungi, plants, insects, snails), and second to their solubility in both organic solvents and water. Thus, PCP can be applied to a wide spectrum of materials. In various concentrations, solvents, and formulations, pentachlorophenols are registered for use on beans (for replanting purposes only), wood, leather, burlap, masonry, cordage, paints, petroleum, pulp and paper mill systems, weeds on seed crops (preharvest desiccant), secondary oil recovery injection waters, and commercial and industrial water cooling towers and evaporation condensers (Na-PCP).

Approximately 50,000,000 pounds of PCP is produced annually in the United States. The major use of PCP in the United States is as a wood preservative. This use consumes approximately 80% of all PCP produced. About 11% of the PCP produced is formulated as Na-PCP and used in the production of pressed and insulation board, and in cooling towers. Approximately 6% is used in pulp and paper mills to control the growth of slime-forming bacteria and fungi in paper production; 3% is used for farm treatment of fence posts, home protection against termites, and as a herbicide and pre-harvest desiccant.

The number of Federally-registered products containing PCP and the number of registrants is given in Table 4. In addition, there are 75 State-registered products formulated by 60 registrants.

TABLE 4. Federal registration of pentachlorophenol 1/

	No. Products	No. Registrants
PCP	578	240
Na-PCP	196	88
K-PCP	5	4
L-PCP	6	2

1/ Data from computerized Registration Division files.

E. Regulatory History

Technical PCP was registered for use as a wood preservative in 1948 by Dow Chemical Company and Monsanto Agricultural Products Company. Subsequently, registrations were granted for a wide variety of uses, including agricultural uses such as pre- or postharvest weed treatment and preharvest dessication of seed crops. Until 1970, regulatory actions against PCP were confined to these kinds of uses. For example, in the "USDA Summary of Registered Agricultural Pesticide Uses" issued on August 31, 1968, PCP was registered as a weed killer on alfalfa, cotton, pineapples, and sugarcane. These registrations were cancelled by Pesticide Regulation (PR) Notices 69-4 (February 1, 1969) and 70-4 (February 26, 1970) when the concept of zero tolerance was abandoned by the Department of Agriculture. Currently, PCP is registered for agricultural use only as a seed treatment for nonfood uses on beans, alfalfa, clover, lespedeza, and vetch.

On September 28, 1970, USDA published PR Notice 70-22. Although PCP was not mentioned by name, the Notice stated "Appropriate regulatory action will be taken under the provisions of the Act (FIFRA) if these chlorodioxins are found in any economic poison." The notice was directed toward the presence of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the herbicides 2,4,5-trichlorophenoxyacetic acid and silvex; PCP and its chlorodioxins were not mentioned. The Dow Chemical Company, however, interpreted the Notice to apply to PCP and took action to reduce chlorodioxin levels in its technical PCP.

At the time the Notice was issued, methodologies for the analysis of dioxins in PCP were not totally reliable. Therefore, there was disagreement within the industry on the actual levels of dioxins in their products. There were no toxicological studies on PCP dioxins, and toxic effects were inferred from chloracne and chick edema effects of commercial PCP. These same effects are caused by TCDD. In the absence of such evidence, regulatory action on dioxin levels in PCP was not possible. This situation continued until 1974, when research involving purified PCP (i.e., relatively free of dioxins), in contrast to commercial PCP, began to appear in the literature.

II. Summary of Scientific Evidence Relating to Rebuttable Presumption Criteria

A. Reproductive and Fetotoxic Effects in Mammalian Species

40 CFR 162.11(a)(3)(ii)(B) provides that "a rebuttable presumption shall arise if a pesticide's ingredient(s)... Produces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety." This section reflects concern that chronic exposure to chemicals may result in injury to the reproductive system and/or the fetus and provides that a rebuttable presumption shall arise if chronic chemical exposure in test animals produces such results, and if human exposure to the chemicals exceed an ample margin of safety.

In the studies summarized below, fetotoxic and teratogenic effects have been reported in rats exposed to purified and commercial grade PCP. These same effects were observed in rats exposed to a mixture of two unspecified isomers of HCDD. Specifically, exposure to PCP and these HCDD isomers resulted in statistically significant increases in the incidence of skeletal and soft tissue anomalies, growth-retarded fetuses and of embryonic resorption in the litters of treated dams.

1. Fetotoxicity

a. Studies with PCP

Schwetz et al. (1974) studied the effects of purified and commercial grade PCP on rat embryonal and fetal development. The compositions of the two types of PCP are given in Table 5. Dosages of 5, 15, 30, and 50 mg/kg/day of both PCP types were administered by gavage on gestation days 6 through 15 inclusive. Their results, shown in Table 6, may be summarized as follows: both purified and commercial PCP caused statistically significant increases in fetal resorptions at the two higher doses, as well as among litters exposed to 15 mg/kg/day of commercial PCP. Specifically, the resorption rate ranged from 27.2% (64/235) to 100% (229/229) in the two highest (commercial and purified) dose groups, compared to 4.2% (15/358) in the control group. At 30 and 50 mg/kg/day, purified PCP had a more pronounced effect than commercial PCP. For example, at 50 mg/kg/day, purified PCP caused 100% fetal resorbtion, while commercial

TABLE 5. Composition of pentachlorophenol materials used by Schwetz et al. (1974)

	Commercial Grade	Purified
Identification	Lot No. MM06210-9822A	Ref. No. 27-91-1
Phenolics, %[1/]		
Pentachlorophenol	88.4	98+
Tetrachlorophenol	4.4	0.27
Trichlorophenol	< 0.1	0.05
Higher Chlorinated Phenoxyphenols	6.2	0.5
Nonphenolics, ppm[2/]		
Dibenzo-p-dioxins		
2,3,7,8-tetrachlorodibenzo-p-dioxin	< 0.05	< 0.05
Hexachlorodibenzo-p-dioxin	4	< 0.5
Heptachlorodibenzo-p-dioxin	125	< 0.5
Octachlorodibenzo-p-dioxin	2500	< 1.0
Dibenzofurans		
Hexachlorodibenzofuran	30	< 0.5
Heptachlorodibenzofuran	80	< 0.5
Octachlorodibenzofuran	80	< 0.5

1/ Determined by gas-liquid chromatography.
2/ Determined by use of an LKB 9000 gas chromatograph-mass spectrometer.

TABLE 6. Effect of pentachlorophenol on the incidence of fetal resorptions and on the sex ratio of survivors[1/]

Test Material and Dose, mg/kg/day	Resorptions: Among Fetuses %	Among Fetuses No.	Among Litters %	Among Litters No.	Sex Ratio M	Sex Ratio F
Vehicle Control[2/]	4.2	15/358	30.3	10/33	50	50
Pentachlorophenol[3/]						
Commercial						
5.8[4/]	7.1	15/212	55.6	10/18	50	50
15	8.8	17/194[5/]	64.7	11/17[5/]	52	48
34.7[6/]	27.2	64/235[5/]	94.7	18/19[5/]	60	40
50	58.1	108/186[5/]	93.3	14/15[5/]	79	21[5/]
Purified						
5	4.2	8/189	46.7	7/15	48	52
15	5.9	13/221	38.9	7/18	50	50
30	97.5	233/239[5/]	100.0	20/20[5/]	83	17[5/]
50	100.0	229/229[5/]	100.0	19/19[5/]	-	-

1/ Adapted from Schwetz et al. (1974)
2/ 2.0 ml/kg body weight corn oil per day.
3/ Dosages administered in 2.0 ml corn oil/kg body weight.
4/ Equivalent to 5.0 mg/kg/day purified PCP.
5/ Indicates values significantly different from control values by the binomial expansion test, $p < 0.05$.
6/ Equivalent to 30.0 mg/kg/day purified PCP.

PCP caused 58% resorption. At 30 (purified) and 50 (commercial) mg/kg/day, there were statistically significant differences in the sex ratio of surviving fetuses; males were heavily predominant. Schwetz et al. found that administration of PCP during early organogenesis (days 8 through 11 of gestation) had more pronounced effects on fetal resorption than did its administration during late organogenesis (days 12 through 15) (Table 7).

The no-effect dose for fetal resorptions was 5.8 commercial grade PCP/kg/day and 15 mg purified PCP/kg/day. Measurements were also taken on fetal body weight and crown-rump length, both of which decreased with the increase in dosage. The no-effect dose for these parameters was 15 mg commercial grade or purified PCP/kg/day.

Larsen et al. (1975) fed 60 mg ^{14}C-PCP/kg body weight to pregnant Charles River CD strain rats on day 15 of gestation. They detected negligible amounts of ^{14}C-PCP in the placentas and fetuses up to 32 hours after dosage. This indicated that the amount of PCP that passes through the placental barrier on day 15 is negligible. In a separate experiment that used unlabeled PCP and was reported in the same paper, single oral doses of 60 mg/kg administered to separate groups of animals on days 8, 9, 10, 11, 12, or 13 of gestation had no statistically significant effect on the rate of resorption of the test animals as compared to controls. However, statistically significant reductions in fetal weight were reported in days 9 and 10.

TABLE 7. Effect of administration of pentachlorophenol during early or late organogenesis on the incidence of fetal resorptions and on the sex ratio of offspring[1/]

Test Material and Dose, mg/kg/day	Resorptions: Among Fetuses %	Among Fetuses No.	Among Litters %	Among Litters No.	Sex Ratio M	Sex Ratio F
DAYS 8-11 OF GESTATION						
Vehicle Control	7.6	13/172	53.3	8/15	53	47
Pentachlorophenol						
Commercial 34.7[2/]	45.3	105/232[3/]	94.7	18/19[3/]	55	45
Purified 30.0	91.1	163/179[3/]	100.0	16/16[3/]	62	38
DAYS 12-15 OF GESTATION						
Vehicle Control	4.2	11/259	40.9	9/22	46	54
Pentachlorophenol						
Commercial 34.7[2/]	6.1	13/213	58.8	10/17	52	48
Purified 30.0	4.5	11/244	45.0	9/20	49	51

1/ Adapted from Schwetz et al. (1974).
2/ Equivalent to 30.0 mg/kg/day purified PCP.
3/ Values significantly different from control values by the binomial expansion test, $p < 0.05$.

Hinkle (1973) reported on the fetotoxic effects of PCP in the Golden Syrian hamster. After doses of 1.25, 2.5, 5, 10, and 20 mg/kg were administered by gavage on days 6 through 10 of gestation, he reported no differences between control and test animals in these parameters: maternal body weight, fetal weight, litter size, and number of resorptions. Increased toxicity was noted at the two highest doses, but these increases were "minimal in number." The author stated that PCP was found in detectable amounts (unspecified) in the untreated animals as well as in their diet.

Fahrig (1978), in a study to be described in more detail in Section III. B., observed decreases in litter size after injection of 50 and 100 mgms PCP/kg body weight into the peritoneal cavity of pregnant mice at day ten of gestation. Control mice, on the average, produced 6.4 fetuses/dam. Litter size was reduced at the 50 mg/kg dose to 3.67 fetuses/dam, and to 3.92 fetuses/dam at the 100 mg/kg dose. PCP was administered in a 10% solution of dimethylformamide; a vehicle control was not reported. Litter size calculations included dams that had no litters.

The Schwetz et al. study cited earlier establishes that ingestion of PCP by pregnant rats during organogenesis produces lethal and toxic effects such as fetal resorption and reduced body size. Comparable effects were not observed when larger but single doses were administered to rats during organogenesis (Larsen, 1975), nor when single doses were administered to hamsters (Hinkle, 1973).

Table 8. Effect of treatment with chlorinated dibenzo-p-dioxin on maternal and fetal body measurements and the incidence of fetal resorption[1/]

Test Compound[2/]	No. litters	Maternal weight gain, g[3/] Days 6-13	Days 13-21	Days 6-21	Fetal body weight,g[4/]	Fetal crown-rump length,m mm	Fetal Resorption, %[4/] Population[5/]	Litter[4/]
Control	30	36 ± 2	101 ± 6	137 ± 8	5.68 ± 0.05	44.5 ± 0.1	7(22/337)	47(14/30)
Hexachlorodibenzo-p-dioxin (4)								
0.1 ug/kg/day	19	28 ± 2	102 ± 5	130 ± 5	5.73 ± 0.04	43.8 ± 0.1	5(10/217)	47(9/19)
1 ug/kg/day	19	27 ± 3	99 ± 5	126 ± 6	5.93 ± 0.16	45.7 ± 0.5	9(20/218)	74(14/19)
10 ug/kg/day	18	22 ± 3[7/]	97 ± 5	119 ± 6	5.12 ± 0.05[7/]	42.6 ± 0.2[7/]	7(57/229)[7/]	94(17/18)[7/]
100 ug/kg/day	19	6 ± 2[7/]	13 ± 7[7/]	19 ± 9[7/]	3.65 ± 0.28[7/]	35.2 ± 0.7[7/]	85(194/227)[7/]	100(19/19)[7/]
Octachlorodibenzo-p-dioxin (5)								
100 mg/kg/day	12	32 ± 2	100 ± 8	131 ± 7	5.73 ± 0.09	43.6 ± 0.4	8(11/131)	42(5/12)
500 mg/kg/day	17	35 ± 3	115 ± 4	150 ± 5	5.69 ± 0.05	44.5 ± 0.2	5(9/199)	41(7/17)

1/ Adapted from Schwetz et al. (1973).
2/ Hexachlorodibenzo-p-dioxin sample: purity = >99%; two unspecified isomers in ratio of 89:11. All samples administered on days 6-15 of gestation as a corn oil:acetone (9:1) solution. Octachlorodibenzo-p-dioxin samples: purity = 98.86%.
3/ Mean ± standard error for various gestation times.
4/ Mean of litter means ± standard error.
5/ % (number resorptions/number implantations).
6/ % (number litters with at least one resorption/number litters).
7/ Significantly different from control by an analysis of variance and Tukey's test or the 2 x 2 contingency table (resorptions), $p < 0.05$.

b. Studies with Dioxins

Schwetz et al. (1973) administered purified hexachloro-p-dibenzodioxins (HCDD) (two unspecified isomers) and octachloro-p-dioxin (OCDD) by gavage to pregnant Sprague-Dawley rats on days 6 through 15 of gestation. Doses were 0.1, 1, 10, or 100 ug/kg/day HCDD and 100 or 500 mg/kg/day OCDD.

The results of these studies are shown in Table 8. For HCDD, there were statistically significant increases over controls in fetal resorptions at the 10 and 100 mg/kg/day doses, as well as decreases in fetal body weight and fetal crown-rump length. In contrast, OCDD at both dose levels (100 and 500 mg/kg/day) produced no fetal resorptions or other effects except for an increase in the incidence of subcutaneous edema at the high dose level.

The HCDD used in this experiment was reported by the authors to have a purity in excess of 99%, and to consist of two isomers in the ratio 89:11. The presence of HCDD in PCP and the low HCDD doses producing fetotoxic effects in pregnant rats strongly suggests that exposure to HCDD via exposure to PCP can cause chronic effects as defined in 40 CFR 162.11(a)(3)(ii)(B).

2. Teratology

a. Studies with PCP

Schwetz et al. (1974a) also investigated the teratogenic effects of PCP on rats. In this study they observed fetal anomalies produced by oral administration (gavage) of 5.8, 15, 34.7, and 50 mg/kg/day of commercial grade PCP, and 5, 15, and 30 mg/kg/day of purified PCP. In one experiment, they administered these amounts of PCP during days 6 through

TABLE 9. Effect of pentachlorophenol on the incidence of fetal anomalies

	Vehicle Control	Pentachlorophenol (mg/kg/day) Commercial Grade 5.8	15	34.7	50	Purified Grade 5	15	30
SOFT TISSUE ANOMALIES No. of Litters	33	18	16	19	13	15	18	2
		Percent of Litters Affected						
Subcutaneous Edema	18	11	50 2/	84 2/	62 2/	0	78 2/	100 2/
Dilated Ureters	0	0	0	21 2/	0.	0	0	0
SKELETAL ANOMALIES No. of Litters	31	18	16	19	12	15	18	2
		Percent of Litters Affected						
Skull (delayed ossification)	19	39	31	37	8	60 2/	72 2/	0
Lumbar Spurs	13	28	88 2/	37 2/	83 2/	20	78 2/	0
Ribs (supernumerary, lumbar, or fused)	0	0	0	95 2/	83 2/	0	33 2/	100 2/
Vertebrae (supernumerary, abnormal shape, delayed ossification, missing or unfused centers of ossification)	19	0	19	95 2/	100 2/	13	78 2/	100 2/
Sternebrae (supernumerary, delayed or unfused centers of ossification, fused or staggered)	16	11	13	89 2/	83 2/	33	39 2/	100 2/

1/ Administered po in corn oil on days 6-15 of gestation. The doses of 5.8 and 34.7 mg commercial grade PCP/kg/day are equivalent to 5 and 30 mg/kg/day purified PCP, respectively.
2/ Incidence significantly different from control (binomial expansion test, $p < 0.05$).

Modified from Schwetz et al. (1974)

TABLE 10. Effect of administration of pentachlorophenol during early or late organogenesis on the incidence of fetal anomalies

Anomalies	Days 8-11 of Gestation: Vehicle Control	Pentachlorophenol 1/ Commercial 34.7 mg/kg/day	Purified 30 mg/kg/day	Days 12-15 of Gestation: Vehicle Control	Pentachlorophenol 1/ Commercial 34.7 mg/kg/day	Purified 30 mg/kg/day
SOFT TISSUE ANOMALIES No. of Litters	15	17	4	22	17	20
	Percent of Litters Affected					
Subcutaneous edema	7	82 2/	100 2/	59	35	95 2/
SKELETAL ANOMALIES No. of Litters	15	18	6	22	17	20
	Percent of Litters Affected					
Skull (delayed ossification)	33	33	17	36	41	70 2/
Ribs (supernumerary or fused)	0	100 2/	100 2/	0	0	0
Vertebrae (supernumerary, abnormal shape, delayed ossification, fused, missing or unfused centers of ossification)	27	100 2/	100 2/	9	0	0
Sternebrae (delayed or unfused centers of ossification, fused or staggered)	47	94 2/	100 2/	50	82 2/	85 2/

1/ Administered po in corn oil during early or late organogenesis. The dose of 34.7 mg/kg/day commercial grade PCP is equivalent to 30 mg/kg/day purified PCP.
2/ Incidence significantly different from control (binomial expansion test, $p < 0.05$).

Adapted from Schwetz et al. (1974)

15 of gestation; statistically significant increases in skeletal defects of the ribs, sternebrae and vertebrae were observed in both treatment groups (Table 9). In a second experiment, they gave 30.0 (purified) and 34.7 (commercial grade) mg/kg/day PCP on days 8 through 11 of gestation to one group of animals, and on days 12 through 15 to a second group; statistically significant increases in abnormal sternebrae and skulls were observed in animals treated with purified PCP and abnormal sternebrae in animals treated with commercial PCP (Table 10).

These results establish that the higher doses of PCP produce fetal anomalies in the rat. At the 30 mg/kg/day dose the effects of purified PCP were more pronounced than those of the equivalent of commercial grade PCP.

b. Studies with Dioxins

In the 1973 paper cited above, Schwetz et al. reported teratogenic effects from the dioxins found in PCP. They administered doses of 0.1, 1, 10, and 100 ug HCDD/kg/day to pregnant Sprague-Dawley rats on days 6 through 15 of gestation. They found statistically significant increases over the controls in all of the teratogenic parameters observed at 100 ug/kg. For example, cleft palate was observed in 47% (8/17) of the fetuses exposed to HCDD, compared to none (0/156) in the controls; 12% (2/17) of the treated fetuses had dilated renal pelvis compared to 0.6% (1/56) in the controls; and 31% (5/16) of the treated fetuses had abnormal vertebrae compared to 6% (9/158) in the controls. Subcutaneous edema was observed at all doses except 0.1 ug/kg/day, which was considered the no-effect dose. In contrast, OCDD did not cause teratogenicity at 100 mg/kg/day; doses of 500 mg/kg/day caused subcutaneous edema, but no other effects.

Table 11 summarizes the results of treatment with HCDD.

3. Exposure Analysis

In order to determine whether a presumption should be issued based on reproductive and fetotoxic effects, pursuant to Section 162.11(a)(3)(ii)(B), the Agency must determine whether or not an ample margin of safety exists between the levels of PCP which produce reproductive and fetotoxic effects, and the level(s) to which the population at risk (women of child-bearing age) can reasonably be anticipated to be exposed.

This section presents estimates of dietary, inhalation and dermal exposure to PCP and HCDD on a "worst case" basis. These estimates are based on the exposure of a pregnant woman in the home and at work at major PCP and Na-PCP use work sites, i.e., wood preserving plants, cooling towers, tanneries, and construction sites. This approach to PCP exposure analysis is taken because of the fetotoxic and teratogenic effects of PCP described in Section II.A.1 and II.A.2 and because of the continued movement of women into all areas of the labor force. Estimates are computed on the basis of 60 kg pregnant woman, 100% absorption via dietary and inhalation exposure, and 10% adsorption via dermal contact for both PCP and Na-PCP. There is no data available on dermal absorption of PCP by pregnant women; the 10% estimate is based on the penetration value of 7-15% of several chlorinated hydrocarbons, reported by Maibach and Feldman (1974). A normal breathing rate is defined as 1.8 m^3/hour at work, and 1.0 m^3/hour in the home.

Inhalation of water vapor containing Na-PCP is an exposure route for workers in industrial cooling towers, paper pulp mills and tanneries. Appendix I shows the calculations used to derive estimates of PCP in water vapors at these sites.

Exposure to HCDD is developed using the same bases as for PCP. In addition these assumptions are made: that the HCDD content of most technical PCP and Na-PCP in the market place is 4 ppm (Table 2) and that assimilation of HCDD is identical to that of PCP and Na-PCP. Using these assumptions, exposure to HCDD is equal to the exposure to PCP times the factor 4×10^{-6} (4 ppm).

a. Dietary Exposure

Although PCP has no registered uses on any food commodity, PCP residues have been measured in foodstuffs (Table 1), and therefore there is exposure to PCP in the diet. Using the data in Table 1 and the average daily intake of the commodities which have been found to contain PCP, it is possible to obtain an estimate of PCP exposure from foodstuffs. Table 12 displays these results, computed for average and maximum PCP intake per day. For a 60 kg pregnant woman, the intake would be:

0.0015 mg/day/60 = 0.025 ug/kg/day average,

or 0.0181 mg/kg/day/60 = 0.302 ug/kg/day maximum.

For HCDD, intake would be:

0.1×10^{-6} ug/kg/day average, or 1.21×10^{-6} ug/kg/day maximum.

b. Dermal Exposure

i. Homeowners

PCP is sold in retail stores for treatment of wood to prevent rot and decay in boats, masonry, fences, and other sites. Common directions call for two coats of 5% PCP in mineral oil or a 3 to 30 minute soak. Assuming that a 60 kg applicator spills enough of the solution to cover one hand (10 ml or about 10 grams), the exposure would be:

(10 gms x 0.05 x 0.1)/60 = 0.833 mg/kg/day.

For HCDD, exposure would be 3.33×10^{-6} mg/kg/day.

TABLE 11. Effect of treatment with hexachlorodibenzo-p-dioxin on the incidence of fetal anomalies 1/

		Incidence of Anomalies After Treatment on Days 6-15 of Gestation				
		Control	0.1 ug/kg/day	1 ug/kg/day	10 ug/kg/day	100 ug/kg/day
SOFT TISSUE ANOMALIES						
Cleft Palate	P 2/	0 (0/156)	1 (1/104)	0 (0/99)	0 (0/86)	47 (8/17) 4/
	L 3/	0 (0/28)	5 (1/19)	0 (0/19)	0 (0/18)	73 (8/11) 4/
Dilated Renal Pelvis	P	0.6 (1/156)	0 (0/104)	2 (2/99)	6 (5/86) 4/	12 (2/17) 4/
	L	4 (1/28)	0 (0/19)	5 (1/19)	17 (3/18)	18 (2/11)
Subcutaneous Edema	P	5 (8/156)	6 (6/104)	55 (54/99) 4/	100 (86/86) 4/	100 (17/17) 4/
	L	21 (6/28)	32 (6/19)	100 (19/19) 4/	100 (18/18) 4/	100 (11/11) 4/
SKELETAL ANOMALIES						
Split Vertebral Centra	P	6 (9/158)	2 (2/103)	1 (1/99)	7 (6/86)	31 (5/16) 4/
	L	19 (5/27)	5 (1/19)	6 (1/18)	29 (5/17)	56 (5/9) 4/
Split Sternebrae	P	0.6 (1/158)	1 (1/103)	2 (2/99)	2 (2/86)	31 (5/16) 4/
	L	4 (1/27)	5 (1/19)	11 (2/18)	12 (2/17)	56 (5/9) 4/
Delayed Ossification of Sternebrae	P	11 (18/158)	28 (29/103)	12 (12/99)	34 (29/86)	56 (9/16) 4/
	L	44 (12/27)	74 (14/19)	50 (9/18)	71 (12/17)	56 (5/9)

1/ Adapted from Schwetz et al. (1973).
2/ Incidence among fetal population: % (number of affected fetuses/number fetuses examined).
3/ Incidence among litters: % (number of affected litters/number litters examined).
4/ Significantly different from control by 2 x 2 contingency table, $p < 0.05$.

Table 12. Estimated Average and Maximum Daily Intake of PCP in the Diet

Commodity	Average Intake kg/day 1/	Average PCP Residue mg/kg 2/	Maximum PCP Residue mg/kg	Average PCP Intake mg/day	Maximum PCP Intake mg/day
Dairy Products	0.477	0.0005	0.01	0.0002	0.0048
Grains and Cereals	0.181	0.001	0.013	0.0002	0.0024
Root Vegetables	0.261	0.001	0.010	0.0003	0.0026
Fruits	0.261	Trace	0.011	-	0.0029
Sugars and Adjuncts	0.135	0.006	0.04	0.0008	0.0054
Totals				0.0015	0.0181

1 From Agricultural Handbook No. 62, August 1961, p. 42

2 From Table 1 (Johnson and Manske, 1977)

ii. Construction Workers

PCP-treated wood is used to construct platforms, fences, porches, and other structures. It is estimated that 6 months after treatment, PCP will be present on the wood surface at about 0.5 mg/ square foot (Koppers Chemical Company, 1978). Dermal exposure occurs if workers handle wood without wearing gloves. Assuming that the worker actually handles wood 40 times during an 8-hour period, and that the area of the hands averages 0.25 sq. ft., then exposure would be:

(40 x 0.5 mg/sq.ft. x 0.25 sq.ft./operation x 0.1)/60 = 8.3 ug/kg/day.

For HCDD, exposure would be 33.2×10^{-6} ug/kg/day.

iii. Cooling Tower Workers

Na-PCP is used to control the growth of algae, bacteria and fungi in water cooling systems. Formulations typically contain 80% Na-PCP, and are added to the system at concentrations of 60 ppm for initial cleaning, then reduced to 30 ppm to maintain control. A worker could be exposed dermally to 100 ml of cooling water containing 80% x 60 ppm formulation, or 48 ppm (48 mg/liter), of Na-PCP. (Shamaiengar, 1978). Exposure would then be:

(48 mg/l x 0.1 liter x 0.1)/60 kg = 0.008 mg/kg/day.

During routine maintenance, this would drop to 0.004 mg Na-PCP/kg/day. For HCDD, exposure would be 0.032×10^{-6} mg/kg/day for initial cleaning, and 0.016×10^{-6} mg/kg/day during routine maintenance.

iv. Paper/Pulp Mill Workers

To control slime, algae and bacteria, Na-PCP is added to the paper-pulp slurry during the manufacturing process. A typical product contains 45% active ingredients of which 25% is Na-PCP. The concentration of the product used is 450 ppm, of which 113 ppm (113 mg/l) is Na-PCP.

During the process, samples of pulp slurry are taken periodically. If a worker takes samples hourly (8 times/day) and wets one hand with 10 ml of the slurry (International Paper Company, 1978), the exposure would be:

$$(8 \times 0.01 \text{ liter} \times 113 \text{ mg/l} \times 0.1)/60 \text{ kg} =$$

$$0.015 \text{ mg PCP/kg/day}$$

For HCDD, exposure would be 0.060×10^{-6} mg/kg/day.

v. Tannery Workers

A typical product used to control growth of slime and fungi contains 12.9% PCP. With a use concentration of 1:4,000, the solution would contain 1 gm/4,000 gm water or 250 ppm of product. If a worker were exposed during normal operations, she could dermally receive about 1400 ml of solution daily on her exposed arms and neck (Shamaiengar, 1978). Exposure here would be:

$$(1.4 \text{ liter} \times 250 \text{ mg/l} \times 0.129 \times 0.1)/60 \text{ kg} =$$

$$0.075 \text{ mg Na-PCP/kg/day}$$

For HCDD, exposure would be 0.3×10^{-6} mg/kg/day.

vi. Workers in Pressure-Treatment Plants

Although data is not available on dermal exposure at these sites, it can be assumed that this exposure is at least equal to exposure at construction sites. Freshly treated wood can be expected to contain more PCP on its surface than wood used at construction sites, the latter having been subjected to weathering, i.e., leaching, vaporization, etc., in the interval between pressure treatment at the plant and use at construction sites. Therefore, dermal exposure at pressure treatment plants is taken to be at least the same as that at construction sites.

c. Inhalation Exposure

i. In the Home

The only data available on the vaporization of PCP from treated wood in an enclosed area is provided by Gebefugl et al. (1976). They found as much as 160 ug PCP/m^3 in the air of a test area treated, according to the label instructions, with paint containing PCP. The concentration of PCP in the paint was not reported.

Using 160 ug PCP/m^3 as a worst case, and assuming that a 60 kg pregnant woman resides in her home an average of 20 hours per day breathing at a rate of 1.0 m^3/hr, the exposure would be (160 ug PCP/m^3 x 1.0 m^3/hr x 20 hr)/60 = 53.3 ug PCP per day.

For HCDD, exposure would be 213.3×10^{-6} ug/kg/day.

ii. Pressure-Treatment Plants and Construction Sites

In February, 1976, the Center for Disease Control, National Institute for Occupational Safety and Health, conducted an environmental survey of a pressure treatment wood preserving plant in Little Rock, Arkansas. This survey included air sampling for PCP at various sites in the plant area. Table 13 shows the results of the analyses of these samples.

The highest value reported, 8 ug/m^3, was in an area where about twice each month 200-250 fifty (50) pound bags of solid PCP were manually opened and dissolved in heated fuel oil. Assuming that, as a worst case, this level of airborne PCP is continuously present throughout the plant,

Table 13.[1/] Pentachlorophenol Concentrations in Air at a Pressure Treatment Plant.
Koppers Company, Incorporated
North Little Rock, Arkansas
February 24, 1976

Sample No.	Operation	Sampling Period (min)	*Type of Sample	**Concentrations (mg/M^3)
A1	Hand Mix Oper.	112	GA	0.004
A3	Hand Mix Oper.	112	GA	0.004
A5	Sampling Man		EQUIPMENT FAILURE - VOID	
A6	Asst. Treater	442	P	0.001
A7	Laborer	445	P	0.001
A8	Laborer	438	P	0.006
A9	Treating Oper.	437	P	0.001
A10	Locomotive Oper.	339	P	<0.001
A2/12	Hand Mixer	110	P	0.008
A4/14	Hand Mix Oper.	112	GA	0.003

American Conference on Governmental Industrial Hygienists, Threshold Limit Value Committee 0.5

* GA - General Area; P - Personal

** mg/M^3 = milligrams of substance per cubic meter of air sampled

1/ Source: Health Hazard Evaluation Determination Report Nol. 75-117-371, DHEW, Center for Disease Control, National Institute for Occupational Safety and Health, Cincinnati, Ohio. March, 1977.

the daily exposure of a 60 kg worker at a moderate breathing rate (1.8 m^3/hr) would be:

$$(8\ ug/m^3 \times 1.8\ m^3/hr \times 8\ hours)/60 = 1.92\ ug/kg/day.$$

For HCDD, exposure would be 7.68×10^{-6} ug/kg/day.

In the absence of data on PCP concentrations in the air at construction sites, these values for PCP and HCDD are taken as worst case estimates at these sites.

iii. Cooling Towers

Some persons working near cooling towers can be exposed by inhalation of vapors and drift containing Na- PCP. The relative amounts of Na-PCP and water evaporating from the cooling water is a function of their molecular weights and vapor pressures, and is estimated to be 0.02 ug PCP/m^3 (Appendix 1). Exposure therefore would be:

$$(0.02\ ug/m^3 \times 1.8\ m^3/hr \times 8\ hr)/60 = 0.004\ ug\ Na\text{-}PCP/kg/day.$$

For HCDD, exposure would be 0.016×10^{-6} ug Na-PCP/kg/day.

Drift is the entrained water carried from the tower by the exhaust air. In an average cooling tower, approximately 1% of the cooling fluid is lost to drift. For a properly functioning cooling tower, the ratio of water to air weight should be unity in order to insure maximum heat exchange. At an average cooling rate of 300 gallons/minute, this weight of water is equivalent to the weight of 880 m^3 of air. The drift (3 gallons or 11.355 liters), distributed in this volume of air, gives an Na-PCP concentration of:

$$(11.355\ l \times 48\ mg/l)/880\ m^3 = 0.6\ mg\ Na\text{-}PCP/m^3.$$

TABLE 14. Estimates of Human Exposure to PCP and HCDD 1/

Site	PCP ug/kg/day				HCDD ug x 10^{-6} /kg/day			
	Dietary	Dermal	Inhalation	Total	Dietary	Dermal	Inhalation	Total
Home	0.025 av	833	53.3	886.32	0.1 av	3330	213.3	3543.40
	0.302 max			886.60	1.21 max			3544.51
Construction Sites	0.025 av	8.33	1.92	10.28	0.1 av	33.2	7.68	40.98
	0.302 max			10.55	1.21 max			42.09
Cooling Towers	0.025 av	8 2/	0.004	158.03 5/	0.1 av	32 2/	0.016	632.02 5/
	0.302 max	3/ 4	4/ 150	5/ 154.31	1.21 max	3/ 16	4/ 600	5/ 617.23
Paper Pulp Mills	0.025 av	15	0.01	15.04	0.1 av	60	0.04	60.14
	0.302 max			15.31	1.21 max			61.25
Tanneries	0.025 av	75	0.003	75.03	0.1 av	300	0.012	300.11
	0.302 max			75.31	1.21 max			301.22
Pressure Treatment Plants	0.025 av	8.33	1.92	10.28	0.1 av	33.2	7.68	40.98
	0.302 max			10.55	1.21 max			42.09

1/ Based on a 60 kg pregnant woman
2/ Initial Treatment
3/ Routine Treatment
4/ Cooling Tower Drift
5/ Inhalation of cooling tower drift + PCP vapors + dermal and dietary exposure

Exposure would then be:

$$(0.6\ mg/m^3 \times 1.8\ m^3/hr \times 8\ hrs)/60 = 0.15\ mg\ Na\text{-}PCP/kg/day$$

For HCDD, exposure would be 0.60×10^{-6} mg/kg/day.

iv. Paper/Pulp Mills

Inhalation exposure is based on a concentration of 0.047 ug Na-PCP/m^3 (see Appendix 1), which computes to:

$$(0.047\ ug/m^3 \times 1.8\ m^3/hr \times 8\ hrs)/60 = 0.01\ ug\ Na\text{-}PCP/kg/day.$$

For HCDD, exposure would be 0.04×10^{-6} ug/kg/day.

v. Tanneries

Inhalation exposure is based on a concentration of 0.013 ug/m^3 (see Appendix 1) so that exposure is:

$$(0.013\ ug/m^3 \times 1.8\ m^3/hr \times 8\ hrs)/60\ kg = 0.003\ ug\ Na\text{-}PCP/kg/day$$

For HCDD, exposure would be 0.012×10^{-6} ug/kg/day.

4. Conclusions

Table 14 summarizes the exposure estimates for PCP and HCDD developed in Section II.A.3. Table 15 gives the ratios of the no effect dose level to total exposure to PCP and HCDD of the major use sites.

The foregoing discussion establishes that PCP and possibly its HCDD contaminants cause teratogenic and fetotoxic effects in test animals. The adverse effects observed among the injured fetuses include distorted sex ratios, increased incidences of resorbed embryos, skeletal anomalies and subcutaneous edema. The no-effect levels are 5.8 mg/kg/day for PCP and 1 ug/kg/day for HCDD. In addition, PCP contains other dioxin contaminants which have not been fully characterized toxicologically, and which vary quantitatively from batch to batch.

Data available to the Agency indicate that the physical and chemical properties of PCP, and its commercial uses and distributions lead to substantial human and environmental exposure to this pesticide. In this regard, several factors are significant:

- Current PCP production approximates 50,000,000 pounds per year and production is expected to increase to 80,000,000 pounds per year in the near future (Josephson, 1977). If PCP is equally distributed among the total U.S. population, assuming 60 kg body weight for each individual exposed, the theoretical exposure potential for each person in the general population is 5 mg/kg/day.

- Although PCP is registered mainly for non-agricultural uses, the chemical is widely distributed in the environment and in human tissues. It is present in the blood and urine of persons not known to be exposed to the pesticide. It has been found in the drinking water of 80% of 108 cities sampled, in rainwater in Hawaii, and at low levels in food commodities such as sugars, root vegetables, and grain and cereal products (Table 1).

- PCP is registered for home and industrial uses. As a result, homeowners, workers using PCP in leather, wood, and paper processing plants, and others using products containing PCP are exposed to the chemical. The Agency estimates that use exposure levels range from approximately 0.9 mg/kg/day for home uses to less than 0.01 mg/kg/day at construction sites (Table 14).

- Homes, tanneries, wood pressure treatment plants, paper pulp mills, and cooling towers are major use sites presenting significant exposure potential, particularly since PCP has been shown to be absorbed through the skin. These uses may involve dermal as well as inhalation contact with the pesticide. Further, since 80% of the annual PCP production is used for wood preservatives, substantial numbers of people who spray, dip, and pressure-treat wood at plants may be exposed to the pesticide. In addition, workers using PCP-treated wood at construction sites can be exposed.

The Agency's pre-RPAR review of pesticide exposure is based on data about the chemical under review, other pesticides, general assumptions, and other relevant information. The Agency recognizes that the exposure data available is sketchy and incomplete. The Agency supplemented this data with reasonable worst case assumptions, which by

Table 15. Ratio of No Effect Dose [1] to Total Exposure Estimates [2] for PCP and HCDD

Site	PCP	HCDD
Home	6.55	282
Construction Sites	557	24073
Cooling Towers	37.2	1602
Paper Pulp Mills	382	16477
Tanneries	77.2	3327
Pressure Treatment Plants	557	24073

1) No Effect Doses are 5.8 mg/ug/day for PCP and 1 ug/kg/day for HCDD.

2) Averages of Total Exposures from Table 14.

their nature are conservative. Nevertheless, uncertainty remains as to the validity of the preliminary exposure estimates derived from the data and the assumptions. Therefore, in determining whether an adequate margin of safety exists, the Agency considers it prudent public policy to utilize greater safety factors for PCP than it would in situations where it had greater confidence in the underlying exposure data and assumptions.

40 CFR 162.11(a)(3)(ii)(B) provides that a rebuttable presumption shall arise if a pesticide produces any "chronic or delayed toxic effect in test animals at any dosage up to a level. . . which is substantially higher than that to which humans can reasonably be anticipated to be

exposed, taking into account ample margins of safety . . . ." PCP produces teratogenic and fetotoxic effects in test animals, and workers using PCP in connection with tannery, home, pressure treatment, paper pulp processing, cooling tower and all other uses may be exposed to PCP, with levels at major use sites ranging from 0.01 to 0.9 mg/kg/day. The Agency has concluded that the difference between these human exposure levels and the no-effect level in test animals may not constitute an ample margin of safety. Accordingly, the Agency has concluded that all PCP registrations exceed this risk criterion.

The Agency invites registrants to provide data and information to confirm, refine, or rebut the information upon which the exposure estimates are based. The Agency will use the new data to determine whether or not the presumption has been rebutted and in assessing the risks which PCP uses may present to health and the environment.

III. Studies Relating to Possible Adverse Effects

This section describes studies on PCP which do not meet the criteria outlined in 40 CFR Section 162.11. It is intended to provide information on other toxic effects of PCP, including those resulting from misuse. With the exception of the discussion of oncogenicity and mutagenicity, the studies presented were chosen as examples of these effects; other reports are available in the scientific literature.

A. Oncogenic Effects in Test Animals

In 1969, Innes et al. administered PCP (Dowicide 7) by gavage to mice at doses of 46.4 mg/kg on days 7-28 of age and at 130 ppm (17 mg/kg/day) in the diet for the following 17 months. They reported that this regime caused no significant increase in tumor incidence in test animals as compared to the control animals. In 1976 Schwetz et al. in an

unpublished study submitted to EPA, reported that dietary regimes of Dowicide EC-7 at 1, 3, 10, and 30 mg/kg/day for 22 and 24 months for male and female rats, respectively, did not increase tumor incidence over control animals. The composition of the PCP products used in these studies was similar to those shown in Table 2.

Boutwell and Bosch (1959) tested a series of phenolic chemicals for their ability to induce skin tumors in mice. In these experiments a simple application of 0.3% dimethylbenzanthracene (DMBA) in benzene was first applied to shaved back skin as an initiator. Subsequently, 20% PCP, the promoter, was applied in benzene to the back skin of each test animal twice weekly for 15 weeks. In this short-term study the investigators reported a survival rate of 82.9% (29/35) in the test animals and a rate of 75% (15/20) in control animals treated with benzene only after the initial exposure to DMBA. The average number of papillomas per survivor in the test group was 0.04, slightly less than the 0.07 observed in the controls; the percent of survivors with papillomas was 4.0% as compared to 7% in the control group.

These papers have been reviewed by the EPA Carcinogen Assessment Group and were found to be negative with respect to oncogenic effects of PCP (Albert, 1978).

B. Mutagenic Effects

Fahrig (1974) induced mitotic gene conversion at the ade 8 and trp 5 loci of *Saccharomyces cerevisiae*. Using a concentration of 0.19 millimoles (50 ppm) PCP in 1% dimethyl sulfoxide for 6 hours, he found 6.62 ade 2 convertants per 10^5 survivors

TABLE 16. Induction of forward mutation, intragenic and intergenic recombination in S. cerevisiae MP-1 in vitro with pentachlorophenol at a treatment time of 3.5 hours[1]

Genetic Alteration	Experiment: # of Experiments	Experiment: Concentration (mg/l)	Experiment: Survival[2]	Experiment: Colonies[3] of Genetically Altered Cells Per Survivor	Control: # of Experiments	Control: Colonies[3] of Genetically Altered Cells Per Survivor	Significance
Mutation	4	400	59 ± 8 (10813)	2.00 ± 0.22 (216)	4	0.61 ± 0.07 (113)	< 0.001
Intergen. Rec.				0.47 ± 0.14 (50)		0.49 ± 0.08 (91)	> 0.8
Intragen. Rec.				5.64 ± 0.45 (609)		2.93 ± 0.10 (542)	< 0.001

1. Adapted from Fahrig et al. (1977)

2. Control Survival = 100%

3. Mutants, Convertants (Intragenic Recombination)/10^7 surviviors, Recombinants (Intergenic Recombination)/10^2 survivors

The numbers in parenthesis give the actual numbers of colonies counted.

(control: 0.45 per 10^5 survivors) and 4.31 trp 5 convertants per 10^5 survivors (control: 0.36 per 10^5 survivors). Fahrig did not report on the statistical reliability of these data.

In a later report, Fahrig (1978) studied the mutagenic properties of certain chlorophenols, including PCP, in yeast and mice ("mammalian spot test"). He used S. cerevisiae MP-1, a diploid multipurpose strain for screening intergenic recombination (mitotic crossing over), intragenic recombination (mitotic gene conversion) and forward mutation. He incubated cell suspensions at 25 C with 400 mg/liter PCP for 3.5 hours. He spread aliquots of the suspensions, approximately $3x10^7$ cells, on solid nutrient-deficient (intragenic recombinants and mutants) or complete (intergenic recombinants) media and incubated at 25 C for 4 and 8 days respectively. Fahrig reported statistically significant ($p<0.001$) increases is foward mutation and mitotic gene conversion. Table 16 contains the results of this experiment.

In the mammalian spot test, he mated females of inbred C57BL/6JHan strain mice to males of rotation bred T-stock. The progeny of this combination are susceptible to color spots in the adult coat if a mutagenic agent is injected into the peritoneal cavity of the dam during the tenth day of fetal development. The incidence of color spots in untreated mice bred as described was 0.1%. A dosage of 50 mg/kg PCP to pregnant mice produced color spots in 0.6% of the progeny; a dosage with 100 mg/kg resulted in a 1.3% incidence of spots. Fahrig did not give the statistical significance of these data.

A geneticist and statistician assigned to EPA have reviewed the Fahrig et al. paper (1978). In the opinion of the geneticist, this study does not provide evidence of the mutagenicity of PCP. This opinion was based on the fact that the experiment has certain shortcomings. Among these are absence of information on the controls as well as on maternal toxicity (Mauer, 1978). Only two animals were affected at each dose level; the EPA statistician has found that this response is not statistically significant at the 0.05 level (Rossi, 1978) using the chi-square test.

For these reasons, the reviewers did not consider the mammalian spot test to be sufficient evidence of mutagenicity, and the criteria of multitest evidence of 40 CFR Section 162.11 (a)(3)(ii)(A) are not met.

It should be noted that mutagenicity tests on PCP were negative with the Ames test (Andersen et al. 1972), the host-mediated assay (Buselmaier et al. 1973), and the sex-linked recessive lethal test on Drosophila (Vogel and Chandler, 1974).

C. Chloracne

Chloracne ("chlorine acne") is a human skin disorder characterized by distention of hair follicles by horny cutaneous tissue, and by a decrease or absence of the sebaceous glands in the area of infection. This condition has been observed in workers in PCP manufacturing plants and wood preserving operations (Baader and Bauer, 1951). Chloracne can arise in these workers weeks or months after exposure, and at first was

thought to be due to PCP itself. However, using the rabbit ear test, Jones et al. showed in 1962 that the acnegenic agent in the herbicide 2,4,5-T was its contaminant TCDD. Further study of the acnegenic effects of pure and commercial grade PCP identified its dioxin contaminants as the causative agent (Johnson et al., 1973).

D. Hepatic Effects

Goldstein et al. (1976) fed pure and technical grade PCP to female Sherman rats for 8 months at dosages of 20, 100, and 500 ppm. Technical PCP produced hepatic porphyria at 100 and 500 ppm, and all doses caused increased hepatic aryl hydrocarbon hydroxylase activity, glucuronyl transferase activity, liver weight, cytochrome P-450, and microsomal heme. N-demethylase activity was not affected. In contrast, pure PCP had no significant effect on these parameters, except for increasing glucuronyl transferase at 500 ppm. Both PCP types decreased the rate of body weight gain at 500 ppm. The technical PCP used contained 8 ppm hexa-, 520 ppm hepta-, and 1380 ppm octachlorodibenzodioxin. Pure PCP contained less than 0.1 ppm each of these contaminants.

Kimbrough and Linder (1975) fed 1000 ppm "relatively pure" PCP and technical PCP to male rats for 3 months. All of the animals were reported to have statistically significant enlargement of the liver when compared to the controls. Histological examination of the liver using the light microscope showed that the rats fed technical PCP had foamy cytoplasm or pronounced vacuolation of the hepatocytes, inclusions, single hepatocellular necrosis, interstitial fibrosis, and a brown pigment in macrophages and Kupffer cells.

Examination with the electron microscope showed an increase in smooth endoplasmic reticulum, many lipid vacuoles, and atypical mitochondria. Livers of rats fed the relatively pure PCP showed enlarged hepatocytes, and many cells contained inclusions in their cytoplasm. Electron microscope studies showed a slight increase in smooth endoplasmic reticulum, atypical mitochondria, and some lipid vacuoles. Livers of control rats were normal. Kimbrough and Linder did not report the amount of dioxin contaminants in the pure and technical PCP which was used in the study.

In 1976, Schwetz et al. observed discoloration of the liver in female rats fed 10 or 30 mg/kg/day PCP. Histological examination revealed pigmented material in the hepatocytes surrounding the central veins with smaller amounts present in the reticuloendothelial cells. Hepatocytes in the centrilobular region also contained pigmented material but were not necrotic. The PCP used in this study was representative of Dow's product and contained approximately 30 ppm total dioxins.

E. Toxicity to Humans

There are reports of deaths caused by industrial or accidental exposure to PCP. In most cases exposure occurred by dermal contact, either to PCP in solution or to materials treated with PCP. Bergner et al. (1965) described five cases of PCP intoxication in Winnipeg in 1963. The one fatality involved a worker in a wood treating plant who, using his bare hands, dipped wood into a vat containing a solution of 4.1% PCP in petroleum solvent. A similar incident had previously been reported in 1952 in France by Truhaut et al. In this case two workers were immersing wooden planks in a 3% aqueous solution of a mixture of 80% Na-PCP and 20% sodium tetrachlorophenate. The workers plunged their hands and forearms into the liquid bath to remove the planks. After 6 days of this work, both men became ill and ultimately died. In all three cases, the initial symptoms of intoxication were profuse sweating and elevated temperatures.

These same symptoms were observed in an incident involving nine neonates in a nursery for newborn infants in St. Louis. Robson et al. (1969) reported these and other symptoms in these infants, two of whom died. Other symptoms reported were increased pulse rate (> 150/minute), hepatomegaly, and respiratory distress. Of the seven survivors, six received exchange blood transfusions, and one received only supportive therapy. Exposure was proven to be via percutaneous absorption of Na-PCP which had been mistakenly used to launder the infants' diapers and bed linens.

In a followup paper, Armstrong et al. (1969) measured PCP levels in samples of these diapers and linens, in autopsy tissues, and in the serum of a surviving infant. They detected the following PCP residues: six diapers, 2.64 to 17.20 mg/100 g; two shirts, 7.38 and 7.90 mg/100 g; two shirt backs, 22.40 and 195 mg/100 g; two crib pads, 4.89 and 178.70 mg/100 g; one mattress pad, 14 mg/100 g; one pillow case, 6.25 mg/100 g; and two muslins, 1.15 and 2.80 mg/100 g. In autopsy tissues, PCP measurements were: kidney, 2.8 mg/100 g; adrenal, 2.7 mg/100 g; heart and blood vessel, 2.1 mg/100 g; fat, 3.4 mg/100 g; and connective tissue, 2.7 mg/100 g. An infant who survived through exchange transfusion, had PCP levels of 11.8 mg/100 ml before, 6.5 mg/100 ml during, and 0.2 mg/ml after the transfusion.

TABLE 17. Toxicity of pentachlorophenol to mammals 1/

Organism (Sex, Strain)	Route of Administration	Dose or Concentration	Reference
Wistar Rats	Oral (0.5% in Stanoflex fuel oil)	27.3 mg/kg	Deichmann et al. (1942)
	Subcutaneous (2% in water)	66.3 mg/kg	Deichmann et al. (1942)
	Oral (1% in olive oil)	77.9 mg/kg	Deichmann et al. (1942)
	Oral (2% in water)	210.6 mg/kg	Deichmann et al. (1942)
Albino Rats (M)	Intraperitoneal	56 mg/kg	Furquharson et al. (1942)
Albino Sprague-Dawley Rats (M,F)	Intraperitoneal 8 °C	620 mg/kg (LD-100)	Keplinger et al. (1969)
	Intraperitoneal 26 °C	420 mg/kg (LD-100)	Keplinger et al. (1969)
	Intraperitoneal 36 °C	120 mg/kg (LD-100)	Keplinger et al. (1969)
Albino Wistar Rats (F)	Percutaneous (dermal) 40% w/v in glycerol formaldehyde	149 mg/kg	Noakes and Sanderson (1969)
Sherman Rats (M,F)	Oral (in peanut oil)	146 mg/kg	Gaines (1969)
(F)	Oral "	175 mg/kg	Gaines (1969)
(M)	Dermal "	320 mg/kg	Gaines (1969)
(F)	Dermal "	330 mg/kg	Gaines (1969)

F. Toxicity to Animals

Toxicological data on PCP are complicated by the presence of varying quantities of tetrachlorophenols, dioxins, and furans in the technical material. Table 17 summarizes the available toxicity data for PCP on various mammalian species.

There are reports of fatalities to farm animals following exposure to PCP. Spencer, in 1957, described the deaths of two Hereford cows within 24 hours after drinking a 5% solution of PCP in kerosene. Blevins (1965) reported on the death of a litter of ten pigs kept in a farrowing house whose floor had been overly treated with PCP dissolved in used crankcase oil. They theorized that the pigs were triply exposed by direct adsorption through the skin, from the milk of the gilt, and through inhalation of PCP "aerosol." The gilt recovered when it was moved outside the farrowing house.

Adelman et al. (1976) established the LC-50's for Na-PCP as 0.21 mg/liter for fathead minnows, and 0.22 mg/liter for goldfish. Holmberg et al. (1972) found that the eel (Anguilla anguilla L) did not survive 5 days of exposure to 0.1 ppm Na-PCP in fresh water. Hanes et al. (1968) found that the LC-50 for coho salmon (Onchorynchus kisutch) is 0.15 mg/liter of K-PCP.

The Public Health Service of DHEW has reports on numerous fish kills from 1964 to 1970 that were caused by effluents from wood treatment plant washing into fresh water. Since 1970, the Pesticide Episode Review System (PERS) has recorded only four incidents involving fish kills. This indicates either that wood preserving practices with effluents have become more stringent, or that incidents are not being reported to PERS.

G. Effects of Tetrachlorophenol

As stated previously, the presence of tetrachlorophenols in all PCP products raises the possibility that adverse effects of PCP could be attributed to these compounds. The toxicological properties of tetrachlorophenols have not been studied extensively. The "Registry of Toxic Effects of Chemical Substances," published by the National Institute of Occupational Safety and Health, DHEW, lists the oral LD_{50} for the rat as 140 mg/kg, both for tetrachlorophenol (presumably mixed isomers) and for 2,3,4,6-tetrachlorophenol.

Schwetz et al. (1974a) evaluated the effects of purified and commercial grade tetrachlorophenol on rat embryonal and fetal development. They fed pregnant Sprague-Dawley rats 10 and 30 mg/kg/day on days 6-15 inclusive of gestation. These doses had no effect on resorptions, fetal body weight, or fetal crown-rump length. At 30 mg/kg/day, there were statistically significant increases in delayed ossification of the skull bones for both tetrachlorophenol types. Schwetz et al. observed subcutaneous edema at 10 mg/kg/day with both compounds, but not with the 30 mg/kg/day doses. Since this effect was not dose-related, the authors speculate that it may have been due to chance.

58

TABLE 17. Toxicity of pentachlorophenol to mammals 1/ (Continued)

Organism (Sex, Strain)	Route of Administration	Dose or Concentration	Reference
Sprague-Dawley Rats (M)	Inhalation 80 ml/min	11.7 mg/kg	Hoben et al (1976)
White Mice	Intraperitoneal	29 mg/kg	Pleskova and Bencze (1959)
	Subcutaneous	63 mg/kg	Pleskova and Bencze (1959)
	Oral	130 mg/kg	Pleskova and Bencze (1959)
	Percutaneous	261 mg/kg	Pleskova and Bencze (1959)
Rabbits	Cutaneous	512.5 mg/kg (LD-100)	McGavack et al. (1941)
	Subcutaneous	275.0 mg/kg (LD-100)	McGavack et al. (1941)
	Intraperitoneal	135.5 mg/kg (LD-100)	McGavack et al. (1941)
	Oral	550.0 mg/kg (LD-100)	McGavack et al. (1941)
Sheep	Oral (5% PCP in sawdust)	120 mg/kg (LD-100)	Harrison (1959)
Calves	Oral (5% PCP in sawdust)	140 mg/kg (LD-100)	Harrison (1959)

1/ All values represent the LD_{50} unless otherwise noted.

Appendix 1

Na-PCP Content in Water Vapor

Na-PCP is registered for use as a slimicide in cooling towers, paper pulp mills and tanneries. In these uses, Na-PCP is normally found in water at concentrations of 48, 113 and 32 mg/liter respectively. The humidity in the work areas at these sites can approach 100%, due to vaporization from the large volumes of water required. Na-PCP can also vaporize from solution, and the amounts of Na-PCP in the vapor can be estimated. In the following calculations, it is assumed that the Na-PCP/water solution is an ideal binary system. The following constants will be required:

At 100% humidity and 20°C,

weight of water vapor = $17.3\ gms/m^3$

vapor pressure of water = 17.54 mm

Vapor pressure of Na-PCP (assuming same volatility as PCP) = 4.7×10^{-4} mm

Molecular weight of water = 18

Molecular weight of Na-PCP = 289

In 1 liter of solution, at 48 mg/l Na-PCP, there are approximately $.048/289 = 1.7 \times 10^{-4}$ moles Na-PCP and 1000 gms/18 = 55.5 moles water. According to Raoult's Law, the partial pressure ($p°$) of each component in a binary system equals the mole fraction M_f (moles of component/total moles present) multiplied by the vapor pressure of the component (VP_c):

$$p° = M_f \times VP_c$$

Then:

$$P° \text{ Na-PCP} = (1.7 \times 10^{-4}/55.5)\ (4.7 \times 10^{-4}) = 1.4 \times 10^{-9} \text{ mm}$$

$$P° \text{ Water} = (55.5/55.5)\ (17.54) = 17.54 \text{ mm}$$

The mole fraction of Na-PCP vapor in the air can be calculated using the formula:

$$\text{mole fraction Na-PCP} = \frac{p° \text{ Na-PCP}}{p° \text{ Water} + p° \text{ Na-PCP}}$$

$$= \frac{1.4 \times 10^{-9}}{17.54 + 1.4 \times 10^{-9}}$$

or $\frac{\text{moles Na-PCP in vapor}}{\text{moles water in vapor}} = 8 \times 10^{-11}$

The moles of water on the vapor is

$$\frac{17.3\ gms/m^3}{18} = 0.96 \text{ moles water}/m^3$$

Therefore

$$\text{moles Na-PCP} = 0.96 \text{ moles water}/m^3 \times 8 \times 10^{-11}$$

$$= 7.7 \times 10^{-11} \text{ moles}/m^3$$

and

$$\text{Grams Na-PCP} = 7.7 \times 10^{-11} \text{ moles}/m^3 \times 298$$

$$= 0.02\ ug/m^3$$

These same calculations apply for vapors in pulp mills and tanneries, and using the concentrations of Na-PCP specified earlier, these calculate to:

Pulp mills: Na-PCP = $0.047\ ug/m^3$

Tanneries: Na-PCP = $0.013\ ug/m^3$

[6560-01-M]

LIST OF REFERENCES[1]

1. Adelman, I. R., L. L. Smith, Jr., and G. D. Siesennop. 1976. Acute toxicity of sodium chloride, pentachlorophenol, Guthion (R), and hexavalent chromium to fathead minnows (*Pimephales promelas*) and goldfish (*Carassius auratus*). J. Fish Res. Board Can. 3(2):203-208. Copyright.

2. Ahlborg, U. G., J. E. Lindgren, and M. Mercier. 1974. Metabolism of pentachlorophenol. Arch. Toxicol. 32:271-281. Copyright.

3. Akitake, H., and K. Kobayashi. 1975. Studies on the metabolism of chlorophenols in fish—III. Isolation and identification of an conjugated PCP excreted by goldfish. Bull. of the Japanese Society of Scientific Fisheries. 41(3):321-327. Copyright.

4. Albert, R. E. 1970. The Carcinogen Assessment Group's Preliminary Report on Pentachlorphenol (PCP). Internal Agency Report.

5. Andersen, K. J., E. G. Leighty, and M. T. Takahashi. 1972. Evaluation of herbicides for possible mutagenic properties. J. Agr. Food Chem. 20(3):649-656. Copyright.

6. Anonymous. 1970. Pentachlorophenol poisoning in the home. Calif. Health 27(12):13. Copyright.

7. Armstrong, R. W., E. R. Eichner, D. E. Klein, W. F. Barthel, J. V. Bennett, V. Jonsson, H. Bruce, and L. E. Loveless. 1969. Pentachlorophenol poinsoning in a nursery for newborn infants. II. Epidemiologic and toxicologic studies. J. Pediatrics. 75(2):317-325. Copyright.

8. Arsenault, R. D. 1976. Pentachlorophenol and contained chlorinated dibenzodioxins in the environment. A study of environmental fate, stability, and significance when used in wood preservation. Published by the American Wood-Preservers Association. Copyright.

9. Baader, H. C. E. W., and H. J. Bauer. 1951. Industrial intoxication due to pentachlorophenol. Ind. Med. Surg. 20(6):286-290. Copyright.

10. Bergner, H., P. Constantinidis, and J. H. Martin. 1965. Industrial pentachlorophenol poisoning in Winnipeg. Canad. Med. Assoc. J. 92:448-451. Copyright.

11. Bevenue, A., J. N. Ogata, and J. W. Hylin. 1972. Organochlorine pesticides in rainwater, Oahuu, Hawaii, 1971-1972. Bull. Environ. Contam. Toxicol. 8(4):238-241. Copyright.

12. Bevenue, A., J. Wilson, L. J. Casarett, and H. W. Klemer. 1967. A survey of pentachlorophenol content in human urine. Bull. Environ. Contam. Toxicol. 2(6):319-332. Copyright.

13. Blevins, D. 1965. Pentachlorophenol poisoning in swine. Vet. Med. Small Anim. Clin. 60:455. Copyright.

14. Boutwell, R. K., and D. K. Bosch. 1959. The tumor-promoting action of phenol and related compounds for mouse skin. Cancer Res. 19:413-424. Copyright.

15. Buhler, D. R., M. E. Rasmusson, and H. S. Nakaue. 1973. Occurence of hexachlorophene and pentachlorophenol in sewage and water. Environ. Sci. Technol. 7(10):929-934. Copyright.

16. Buselmaier, W., G. Rohrborn, and P. Propping. 1972. Mutagenicity studies with pesticides by host-mediated assay and the dominant letholity test on the mouse. Biol. Zbe. 91:311. Copyright.

17. Buser, H., and H. P. Bosshardt. 1976. Determination of polychlorinated dibenzo-p-dioxins and dibenzofurans in commercial pentachlorophenols by combined gas chromatography-mass spectrometry. Jour. of the ADAC 59(3):562-569.

18. Casarett, L. J., A. Bevenue, W. L. Yauger, Jr., and S. A. Whalen. 1969. Observations on pentachlorophenol in human blood and urine. Am. Ind. Hyg. Assoc. J. 30:360-366.

19. Choi, J., and S. Aomine. 1972. Effects of the soil on the activity of pentachlorophenol. Soil Sci. Plant Nutr. 18(6):255-260. Copyright.

20. Choi, J., and S. Aomine. 1974a. Adsorption of pentachlorophenol by soils. Soil Sci. Plant Nutr. 20(2):135-144. Copyright.

21. Choi, J., and S. Aomine. 1974b. Mechanisms of pentachlorophenol adsorption by soils. Soil. Sci. Plant Nutr. 20(4):371-379. Copyright.

22. Chu, J. P., and E.J. Kirsch. 1972. Metabolism of pentachlorophenol by an axenic bacterial culture. Appl. Microbiol. 23(5):1033-1035. Copyright.

23. Cranmer, M., and J. Freal. 1970. Gas Chromatographic analysis of pentachlorophenol in human urine by formation of alkyl ethers. Life Sciences 9 (II):121-128.

24. Deichmann, W., W. Machle, K. V. Kitzmiller, and G. Thomas. 1942. Acute and chronic effects of pentachlorophenol and sodium pentachlorophenate upon experimental animals. J. Pharm. Exper. Therp. 76:104-117. Copyright.

25. Dougherty, R. C., and K. Piotrowska. 1976. Screening by negative chemical ionization mass spectrometry for environmental contamination with toxic residues: application to human urines. Proc. Natl. Acad. Sci. USA 73(6): 1777-1781. Copyright.

26. Duncan, C. G., and F. J. Deverall. 1964. Degradation of wood preservatives by fungi. Appl. Microbiol. 12(1):57-62. Copyright.

27. Fahrig, R. 1974. Comparative mutagenicity studies with pesticides. IARC Scientific Publications No. 10:161-181. Copyright.

28. Fahrig, R., Nilsson, C., and Rappe, C. 1978. Genetic activity of chlorophenols and chlorophenol impurities. In Pentachlorophenol, Plenum Press, New York and London.

29. Farquharson, M. E., J. C. Gage and J. Northover. 1958. The biological action of chlorophenols. Brit. J. Pharmacol.13:20-24. Copyright.

30. Ferguson, E. R. 1959. Wood treated with penta can damage pine nursery seedlings. Tree Planters Notes. 38:21-22. Copyright.

31. Firestone, D. 1977. Determination of polychlorodibenzo-p-dioxins and polychlorodibenzofurans in commercial gelatins by gas-liquid chromatography. J. Agric. Food Chem. 25(6):1274-1280. Copyright.

32. Fountaine, J. E., P. B. Joshipura, and P. N. Keliher. 1976. Some observations regarding pentachlorophenol levels in Haverford Township, Pennsylvania. Water Res. 10:185-188. Copyright.

33. Gaines, T. B. 1969. Acute toxicity of pesticides. Toxicol. Appl. Pharmacol. 14:515-534. Copyright.

34. Gebefugl, I., H. Parlar, and F. Korte. 1976. Contributions to ecological chemistry CXXVI. Short note on the analytical determination of pentachlorophenol in closed areas. Chemosphere. 5(4):227-230. Copyright.

35. Goldstein, J. A., M. Friesen R. E. Linder, P. Hickman, J. R. Hass, and H. Bergman. Effects of pentachlorophenol on hepatic drug-metabolizing enzymes and porphyria related to contamination with chlorinated dibenzo-p-dioxins. Unpublished. 33 pp.

36. Hanes, D.,H. Krueger, I. Tinsley, and C. Bond. 1968. Influence of pentahclorophenol on fatty acids of coho salmon (*Onchorynchus kisusci*). Proc. Western Pharmacol. Soc. 11:121-125. Copyright.

37. Harrision, D. L. 1959. The toxicity of wood preservatives to stock. N. Zeal. and Veterinary J. 7:89-94. Copyright.

38. Hetrick, L. A. 1952. the comparative toxicity of some organic insecticides as termite soil poisons. J. Econ. Entomol. 45(2):235-237. Copyright.

39. Hinkle, D. K. 1973. Fetotoxic affects of PCP in the golden Syrian hamster. Unpublished. 8 pp. Given at the 12 annual meeting of the Society of Toxicolgists.

40. Hoben, H. J., S. A. Ching, and L. J. Casarett. 1976. A study of inhalation of pentachlorophenol by rats. III. Inhalation toxicity study. Bull. Environ. Contam. Toxicol. 15(4):463-465. Copyright.

41. Holmberg, B., S. Jensen, A. Larsson, K. Lewander, and M. Olsson. 1972. Metabolic effects of technical pentachlorophenol. (PCP) on the eel *Anguilla anguilla L.* Comp. Biochem. Physiol. 43B:171-183. Copyright.

42. Ide, A., Y. Niki, F. Sakamoto, I. Watanabe, and H. Watanabe. 1972. Decomposition of pentachlorophenol in paddy soil. Agr. Biol. Chem. 36(11):1937-1944. Copyright.

43. Innes, J. R. M., B. M. Ulland, M. G. Valerio, L. Petrucelli, L. Fishbein, E. R. Hart, A. J. Pallotta, R. R. Bates, H. L. Falk, J. J. Gart, M. Klein, I. Mitchell, and J. Peters. 1969. Bioassay of pesticides and industrial chemicals for tumorgenicity in mice: a preliminary note. J. Nat. Cancer. Inst. 42:1101-1114.

44. Johnson, R. D., and D. D. Manske. 1977. Pesticide and other chemical residues in total diet samples (XI). Pest. Mon. Jour. 11(3):116-131.

45. Johnson, R. L., P. J. Gehring, R. J. Kociba, and B. A. Schwetz. 1973. Chlorinated dibenzodioxins and pentachlorophenol. Environ. Health Perspect. 5:171-175.

46. Jones, E. L., and J. Drizek. 1962. A technic for testing acnegenic potency in rabbits, applied to the potent acnegen, 2,3,7,8-Tetra-chlorodibenzo-p-dioxin. Jour. Invest. Dermat. 39:511-517.

47. Josephson, H. R. 1977. Economic, social and environmental benefits from use of wood preservatives. Report to the American Wood Preservers Institute.

48. Keplinger, M. L., G. E. Lanier, and W. B. Deichmann. 1959. Effects of environmental temperature on the acute toxicity of a number of compounds in rats. Toxicol. Appl. Pharmacol. 1:156-161. Copyright.

49. Kimbrough, R. D., and R. E. Linder. 1975. The effect of technical and 99 percent pure pentachlorophenol on the rat liver. Light microscopy and ultrastructure. Abstract. Fourteenth annual meeting of the society of toxicologists. Copyright.

50. Kobayashi, K., H. Akitake, and T. Tomiyama. 1970. Studies on the metabolism of pentachlorophenate, a herbicide, in aquatic organisms—II. Biochemical change of PCP in sea water by detoxication mechanism of Tapes Phillippinarium. Jap. Soc. Sci. Fisheries. Bulletin 36(1):96-102.

[1]Documents marked "Copyright" are available for public inspection in the Office of Special Pesticide Reviews but may not be copied or distributed by the Agency. Papers marked "confidential" may not be copied or distributed.

51. Kutz, R. 1977. Personal communication to D. Cirelli.

52. Kuwatsuka, S., and M. Igarashi. 1975. Degradation of PCP in soils. II. The relationship between the degradation of PCP and the properties of soils, and the identification of the degradation products of PCP. Soil Sci. Plant Nutr. 21(4):405-414. Copyright.

53. Larsen, R. V., G. S. Born, W. V. Kessler, S. M. Shaw, and D. C. van Sickle. 1975. Placental transfer and teratology of pentachlorophenol in rats. Environ. Letters. 10(2):121-128. Copyright.

54. Larsen, R. V., L. E. Kirsch, S. M. Shaw, J. E. Christian, and G. S. Born. 1972. Excretion and tissue distribution of uniformly labeled 14C-pentachlorophenol in rats. J. Pharm. Sci. 61(12):2004-2006. Copyright.

55. Lui, H., and G. D. Sweeney. 1975. Hepatic metabolism of hexachlorobenzene in rats. FEBS Letters. 5(1):225-226.

56. Maibach, H. I., and R. Feldman. 1974. Systemic absorption of pesticides through the skin of man. Report to the Federal Working Group on Pest Management from the Task Group on Occupational Exposure to Pesticides.

57. Mauer, I. 1978. Internal EPA memorandum.

58. McConnell, E. E., J. A. Moore, J. K. Haseman, and M. W. Harris. The comparative toxicity of chlorinated dibenzo-p-dioxin isomers in mice and guinea pigs. Unpublished. 59 pp.

59. McGavack, T. H., L. J. Boyd, F. V. Piccione, and R. Terranova. 1941. Acute and chronic intoxications with sodium pentachlorophenate in rabbits. J. Ind. Hyg. Toxicol. 23:239-251. Copyright.

60. Morton, H. L., J. L. Stewart, and G. P. Bruneau. 1969. Isolation of microorganisms from preservative-treated wood. Forest Products J. 19(1):38-41. Copyright.

61. Noakes, D. N., and D. M. Sanderson. 1969. A method for determining the dermal toxicity of pesticides. Brit. J. Indus. Med. 26:59-64. Copyright.

62. Palmer, C. M., and T. E. Maloney. 1955. Preliminary screening for potential algicides. Ohio J. Sci. 55(1):1-8. Copyright.

63. Pesticide Episode Response Branch, Operations Division, Office of Pesticide Programs, Environmental Protection Agency. September 14, 1976. Summary of Reported Episodes Involving Pentachlorophenol. Pesticide Episode Review Systems Report No. 60. 21 pp.

64. Pleskova, A., and K. Bencze. 1959. Toxic properties of pentachlorophenol. Prac. Lek. 11:348-354.

65. Robson, A. M., J. M. Kissane, N. H. Elvick, and L. Pundavela. 1969. Pentachlorophenol poisoning in a nursery for newborn infants. I. Clinical features and treatment. J. Pediatrics. 75(2):309-316. Copyright.

66. Rossi, L. 1978. Internal EPA memorandum.

67. Schwetz, B. A., P. A. Keeler, and P. J. Gehring. 1974. The effect of purified and commercial grade pentachlorophenol on rat embryonal and fetal development. Tox. Appl. Pharm. 28:151-161. Copyright.

68. Schwetz. B. A., P. A. Keeler, and P. J. Gehring. 1974a. Effect of purified and commercial grade tetrachlorophenol on rat embryonal and fetal development. Tox. Appl. Pharm. 28:146-150.

69. Schwetz, B. A., J. M. Norris, G. L. Sparschu, V. K. Rowe, P. J. Gehring, J. L. Emerson, and C. G. Gerbig. 1973. Toxicology of chlorinated dibenzo-p-dioxins. Environ. Health Perspect. 5:87-99. Copyright.

70. Schwetz, B. A., J. F. Quast, C. G. Humiston, C. E. Wade, G. C. Jersey, R. W. Lisowe, and R. J. Kociba. 1976. Results of a toxicological evaluation of pentachlorophenol sample XD-8108.OOL administered to rats by the dietary route on a chronic basis. Unpublished. 94 pp.

71. Shafik, T. M. 1973. The determination of pentachlorophenol and hexachlorophene in human adipose tissue. Bull. Environ. Contam. Toxicol. 10(1):57-63. Copyright.

72. Spencer, G. R. 1957. Poisoning of cattle by pentachlorophenol in kerosene. J. Amer. Vet. Med. Assoc. 130:299-300. Copyright.

73. Tashiro, S., T. Sasamoto, T. Aikawa, S. Tokunaga, E. Taniguichi, and M. Eto. 1970. Metabolism of pentachlorophenol in mammals. Nippon Noger Kagalar Kaishi. 44:124-129. Copyright.

74. Truhaut, R., P. L'Epee, and E. Boussemart. 1952. Research on the toxicology of pentachlorophenol II. Occupational poisoning in the wood industry: Two fatal cases. Arch. Mal. Prof. 13(6):567-569. Copyright.

75. Vermeer, K., R. W. Risebrough, A. L. Spaans, and L. M. Reynolds. 1974. Pesticide effects on fishes and birds in rice fields of Surinam, South America. Environ. Pollut. 7:217-236.

76. Vogel, E., and J. L. R. Chandler. 1974. Mutagenicity testing of cyclamate and some pesticides in *Drosophila melanogaster*. Speciala 30:621-623. Copyright.

77. Watanabe, I. 1973. Isolation of pentachlorophenol decomposing bacteria from soil. Soil Sci. Plant Nutr. 19(2):109-116. Copyright.

78. Young, H. C., and J. C. Carroll. 1951. The decomposition of pentachlorophenol when applied as a residual pre-emergence herbicide. Agron. J. 43:504-507. Copyright.

**** PRODUCT SEARCH LISTING **** PAGE 1

[6560-01-C]

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000004 BONIDE CHEMICAL CO INC
2 WURZ AVE.
YORKVILLE NY 13495

********** PRODUCT NAME **********

**00188* BONIDE PENTIDE TECHNICAL PENTACHLOROPHENOL 5 % WOOD PRES TERMITE R

****

REGISTRANT *NAME AND ADDRESS*

* 000005 EMPIRE INTERNATIONAL
P.O. BOX 29805
ATLANTA, GA 3[illegible]359

********** PRODUCT NAME **********

**00034* EM 0615

****

REGISTRANT *NAME AND ADDRESS*

* 000043 CABOT SAMUEL INC
ONE UNION ST
BOSTON MA 021 8

********** PRODUCT NAME **********

**00027* CABOTS PENTA WOOD SEAL NO. 3110

**00028* CABOTS BAR DK 3000 CLEAR WOOD PRESERVATIVE

**00071* CABOT'S CONSERVO #3300

**** PRODUCT SEARCH LISTING **** PAGE 2

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000061 KOPPERS COMPANY, INCORPORATED
GENERAL OFFICES KOOPERS BLD
PITTSBURGH PA 15219

********** PRODUCT NAME **********

**00124* KOPPERS WOLMAN SALTS FMP

**00125* KOPPERS WOOD PECKER REPELLENT

**00126* KOPPERS WOODPECKER HOLE FILLER

**00159* BROLITE Z SPAR S 121 ROT INHIBITIVE SEALER GREEN

****

REGISTRANT *NAME AND ADDRESS*

* 000100 CIBA GEIGY CORP
AGRICULTURAL DIV
P.O. BOX 11422
GREENSBORO, NC 27409

********** PRODUCT NAME **********

**00505* PRAMITOL DP CONCENTRATE NO. 3

**00506* PRAMITOL DP CONCENTRATE NO 2

**00507* PRAMITOL DP CONCENTRATE NO 1

**00520* ON TRACK WE 1 HERB/WD CNTR ON R.R RGTS/WY INDUST SITES

**00578* CONQUER PLUS LIQUID VEGETATION KILLER

**00582* CONQUER LIQUID VEGETATION KILLER

**** PRODUCT SEARCH LISTING **** PAGE 3

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT	*NAME AND ADDRESS*
* 000148	THOMPSON HAYWARD CHEMICAL COMPANY BOX 2383 KANSAS CITY KS 66110

********** PRODUCT NAME **********

**00186* DE PESTER PERMAGARD CONC NO. 10

**00425* T H PENTA KIL CONCENTRATE

**00809* DE PESTER PENTA MR 5% PENTA

**00921* SPRA GARD WEED KILLER

****

REGISTRANT	*NAME AND ADDRESS*
* 000168	WASATCH CHEMICAL DIVISION ENTRADA IND INC P O BOX 6219 SALT LAKE CITY UT 84106

********** PRODUCT NAME **********

**00022* WASCO DURACIDE CONCENTRATE

**00024* WASCO PAINT OVER DURACIDE

**00310* MORGRO LIQUID WEED & GRASS KILLER

****

REGISTRANT	*NAME AND ADDRESS*
* 000179	BOGIE R H COMPANY BOX 588 ALEXANDRIA VA 22300

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 4

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 000179

**00061* BO RID BROMOIL PD 2 5 LIQUID WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000237 MUSHROOM SPLY COMPANY
TOUGHKENAMON PA 19374

********** PRODUCT NAME **********

**00008* MSCO FUNGICIDE 'VX'

****

REGISTRANT *NAME AND ADDRESS*

* 000239 CHEVRON CHEMICAL COMPANY
ORTHO DIVISION 940 HENSLEY WAY
RICHMOND CA 94801

********** PRODUCT NAME **********

**01869* TRIOX LIQUID VEGETATION KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000279 FMC CORP.
AGRICULTURAL CHEM DIV
2000 MARKET STREET
PHILADELPHIA, PA 19103

********** PRODUCT NAME **********

**02530* NIAGARA PENTAPHENATE 79 DORMANT FUNGICIDE

**02544* NIAGARA PENTA CONCENTRATE 40

**** PRODUCT SEARCH LISTING **** PAGE 5

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000334 HYSAN CORPORATION
919 W 38TH ST
CHICAGO IL 60609

********** PRODUCT NAME **********

**00245* SUPER NO GROW WEED KILLER

**00247* GREEN KILL HERBICIDE

**00419* GUTTS COMPLETE VEGETATION KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000337 LESTER LABS INC
2370 LAWRENCE ST EAST POINT BRANCH
ATLANTA GA 30344

********** PRODUCT NAME **********

**00021* PENTACHLOR

**00036* LESTER ALGAECIDE B

**00037* LESTER WATERENE

**00059* LESTER ALGAECIDE C

****

REGISTRANT *NAME AND ADDRESS*

* 000341 ZEHRING CHEMICAL COMPANY
2201 NW 20TH AVE
PORTLAND OR 97209

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 6

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 000341

**00037* ZEHRUNG PENTASEAL

**00038* ZEHRUNG PENTGARD WOOD PRESERVATIVE 10 1 CONCENTRATE

**00039* ZEHRUNG PENTASEAL 3 TO 1 CONCENTRATE

**00040* ZEHRING PAINTABLE PENTAGARD WOOD PRESERVATIVE READY TO USE

****

REGISTRANT *NAME AND ADDRESS*

* 000358 NOTT MANUFACTURING COMPANY INC
PLEASANT VALLEY NY 12569

********** PRODUCT NAME **********

**00117* WEED NOT SPRAY

****

REGISTRANT *NAME AND ADDRESS*

* 000363 COOPERS CREEK CHEMICAL CORP
RIVER ROAD
WEST CONSHOHOCKEN, PA 19428

********** PRODUCT NAME **********

**00045* 4 C BRAND DECAYS NOT CLEAR WOOD PENTA PRESERVER

*** PRODUCT SEARCH LISTING *** PAGE 7

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000374 ELRCY TURPENTINE COMPANY
BOX 688
SWAINSBORO GA 30401

********** PRODUCT NAME **********

**00006* PERMATEX PRESERVATIVE FCR WOCD

****

REGISTRANT *NAME AND ADDRESS*

* 000402 HILL MANUFACTURING COMPANY
1500 JONESBORO RD SE
ATLANTA GA 30315

********** PRODUCT NAME **********

**00098* HILCO X WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000413 BARTELS & SHORES CHEMICAL COMPANY
1400 02 ST LOUIS AVE
KANSAS CITY, MO 64101

********** PRODUCT NAME **********

**00057* PIONEER BRAND PENTA 5

**00063* PIONEER BRAND PENTA 4

**** PRODUCT SEARCH LISTING **** PAGE 8

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000449 TECENE CORPORATION
C/O REGULATORY AFFAIRS DEPT, FARMLAND IND , INC
P. O. BOX 7305
KANSAS CITY, MO 64116

********** PRODUCT NAME **********

**00091* JACK FROST WOOD PRESERVATIVE

**00231* WOODBURY PENTA 10 1 WOOD PRESERVATIVE

**00416* SURE DEATH BRAND PCP 10

****

REGISTRANT *NAME AND ADDRESS*

* 000453 WOOD TREATING CHEMICALS DEPT
KOPPERS CO INC
DEPT. KOPPERS CO. 5137 SOUTHWEST AVE.
ST LOUIS MO 63110

********** PRODUCT NAME **********

**00008* TIMBER TOX WD PERS. # 40 CONCNETRATE PENTCNTRT.

**00 174 TIMBERTOX D5 READY TO USE

**00038* B WOOD PRESERVATIVE SPEC AT 7104

**00041* WOODTOX WATER REPELLENT & PENTA WOOD PRESERVATIVE

**00066* PENTA WOOD PRESERVATIVE READY TO USE

**00072* WOOD TREATING SUPER NOXTANE

**00073* READY TO USE WOOD TOX 152 WATER REPELLENT WOOD PRESERVATIVE BROWN

**00074* WOODTREAT AA PENETRATING PENTA COATING

**00083* PRESSTREAT

**00089* POLE TREAT PENETRATING PRESERVATIVE COATING

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 000453

**00103* POLE LIFE PRESERVATIVE PASTE T B

**00140* WDTOX # 109 R RDY TO USE WTR REPELLENT & WD PRESRTVE

**00150* LUMBRELLA 15 YELLOW

**00158* C WOOD PRESERVATIVE AT 8242

**00171* POLE TREAT 15 PENETRATING PRESERVATIVE COATING

**00174* PENTA STAIN 506 REDWOOD

**00175* PENTA STAIN 501 AZTEC BLUE

**00176* PENTA STAIN 502 DRK WALNUT

**00177* PENTA STAIN 503 SUN DANCE YELLOW

**00178* PENTA STAIN 505 PRAIRIE GREEN

**00179* PENTA STAIN 507 CAPE COD RED

**00180* PENTA STAIN 504 TEAL GREEN

**00181* PENTA STAIN 508 DRIFTWOOD

**00182* TRITOX PRESERVATIVE PASTE

**00190* POLE AID NO. 3

**00196* WOODTOX PRE PRIME CONCENTRATE

**00202* WOODTOX PRE PRIME

**00214* COMPOSITION A WOOD PRESERVATIVE WATER REPELLENT

**00215* NO. 140 CONCENTRATE WOODTOX WATER REPELLENT AND WOOD PRESERVATIVE

**00216* NO. 140 READY TO USE WOODTOX WATER REPELLENT AND WOOD PRESERVATIVE

**00219* PENTA WOOD PRESERVATIVE 1 10 CONCENTRATE

**00222* KOPPERS WOOD PRESERVATIVE SURFACE SEMING COMPOUND

**00229* LIQUID NOXTANE 120

**00240* LIQUID NOXTANE SS

**** PRODUCT SEARCH LISTING **** PAGE 10

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 000453

**00245* SUPER DIPRITE WOOD PRESERVATIVE

**00251* WOOD PRESERVATIVE SURFACE SEALING COMPOUND

**00254* LIQUID NOXTANE 1 WOOD FUNGICIDE

**00255* LUMBRELLA 23 WATER REPELLENT PROTECTIVE COATING

**00256* NO. 509 AMBER PENTA STAIN

**00258* LUBRELLA 9 RED BROWN WATER REPELLENT WOOD PRESERVATIVE

**00259* POLE LIFE PRESRVATIVE PASTE TF

**00261* LIQUID NOXTANE 551 WOOD FUNGICIDE

**00262* WOODTOX S CONCENTRATE

**00264* KOPPER'S TIMBERTOX 7 1/2% WOOD PRESERVATIVE

**00265* PRESSTREAT T WOOD PRESERVATIVE

**00266* PENTA STAIN BASE CONCENTRATE

**00267* WOODTOX 140 T CONCENTRATE

**00268* TIMBERTOX 20NW WOOD PRESERVATIVE

**00269* WOODTOX PRE PRIME T CONCENTRATE

**00270* WOODTOX PRE PRIMET

**00271* WOODTOX 140 T

****

REGISTRANT *NAME AND ADDRESS*

* 000456 CHEMICAL FORMULATORS INC
2045 PEACHTREE RD.N.E.
ATLANTA, GEORGIA 30309

********** PRODUCT NAME **********

**00072* CHEMFORM ANTI STAIN

*** PRODUCT SEARCH LISTING *** PAGE 11

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000458 STANDARD TAR PRODUCTS COMPANY
2456 W CORNELL ST
MILWAUKEE WI 53209

********** PRODUCT NAME **********

**00013* USOL CLEAR GRAIN NATURAL WOOD FINISH AND WOOD PRESERVATIVE

**00015* USOL PENTA WOOD PRESERVER

**00016* USOL PENTA TEN TO ONE CONCENTRATE

**00018* REDWOOD PENTA WATER REPELLENT WOOD PRESERVATIVE

**00019* USOL CLEAR PENTA WATER REPELLENT WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 000464 DOW CHEMICAL U S A
PO BOX 1706
MIDLAND MI 48640

********** PRODUCT NAME **********

**00125* DOWICIDE G

**00380* DOW DOWICIDE G ST BEADS SODIUM PENTACHLOROPHENATE TECHNICAL

**00388* DOWICIDE 7 PRILLED PENTACHLOROPHENOL TECHNICAL

**00431* DOWICIDE EC 7

**** PRODUCT SEARCH LISTING **** PAGE 12

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000486 PERCLIN COMPANY, INC
112 W 37TH ST
CHICAGO IL 60609

********** PRODUCT NAME **********

**00003* PEROLIN ALGAE PREVENTIVE #905 BRIQUETTES

****

REGISTRANT *NAME AND ADDRESS*

* 000491 SELIG CHEMICAL INDUSTRIES THE
DIV NATIONAL SERVICE INDS INC PO BOX 43106
ATLANTA GA 30336

********** PRODUCT NAME **********

**00198* SELIG'S MISTER ZERO

****

REGISTRANT *NAME AND ADDRESS*

* 000522 CHEVRON RESEARCH CO.,
A STANDARD OIL CO OF CA. SUBS
575 MARKET STREET
SAN FRANCISCO, CA 94105

********** PRODUCT NAME **********

**00029* CHEVRON WOOD PRESERVATIVE (RF)

*** PRODUCT SEARCH LISTING *** PAGE 13

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000524 MONSANTO COMPANY
AGRICULTURAL PRODUCTS
800 N. LINDBERGH BLVD.
ST LOUIS, MO 63166

********** PRODUCT NAME **********

**00005* MONSANTO PENTA

**00023* SANTOBRITE TECHNICAL SODIUM PENTACHLOROPHENATE NEUTRAL BRIQUETTES

**00055* SANTOBRITE S

**00142* SANTOBRITE NUMBER 2 FINES

**00305* SANTOBRITE PELLETS

****

REGISTRANT *NAME AND ADDRESS*

* 000527 MIDLAND LABS
210 220 JONES ST
DUBUQUE IA 52003

********** PRODUCT NAME **********

**00074* NIX WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000528 AMSCO DIVISION UNION OIL COMPANY OF CALIFORNIA
825 SE THORNTON
MINNEAPOLIS MN 55414

********** PRODUCT NAME **********

**00051* PENTA SEAL CONCENTRATE 1 5

**** PRODUCT SEARCH LISTING **** PAGE 14

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 000528

**00052* AMSCO'S PENTAL SEAL READY TO USE WATER REPELLENT WOOD PRESERVER

**00055* AMSCOS PENTA TREAT READY TO USE WOOD PRESERVER

**00056* PENTA TREAT 1 10 CONCENTRATE WOOD PRESERVER

**00057* BARCOSOTE CREOSOTE PENTA WOOD PRESERVER

****

REGISTRANT *NAME AND ADDRESS*

* 00 538 SCOTT O M & SONS COMPANY
MARYSVILLE OH 43040

********** PRODUCT NAME **********

**00130* TURF BUILDER PLUS MOSS CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 000539 SEARS ROEBUCK & COMPANY
SEARS TOWER DEPT. 766/68TH FLOOR
CHICAGO IL 6 684

********** PRODUCT NAME **********

**00285* SEARS PENTA WOOD PRESERVER

*** PRODUCT SEARCH LISTING *** PAGE 15

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000541 PURITAN CHEM COMPANY
PO BOX 93247 MARTECH STATION
ATLANTA GA 30318

********** PRODUCT NAME **********

**00193* PURITAN #3820 TOTAL WEED KILLER

**00194* PURITAN 3925 TOTAL WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 000548 RILEY BROS INC
860 WASHINGTON
BURLINGTON IA 52601

********** PRODUCT NAME **********

**00022* RILEY BROS 5% PENTA PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 000550 VAN WATERS & ROGERS DIV OF UNIVAR
2256 JUNCTION AVENUE
SAN JOSE, CA 95131

********** PRODUCT NAME **********

**00029* DOWICIDE #7

**00060* GUARDSMAN PENTA WATER REPELLENT WOOD PRESERVATIVE SOLUTION 3:1 CONC

**00092* VW & R STAIN CONTROL

**00101* GUARDSMAN PENTA CONCENTRATE

**** PRODUCT SEARCH LISTING **** PAGE 16

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 000557

**00119* NAMCO PENTACHLOROPHENOL SOLUTION

**00126* MACLEAN'S 40% PENTA

****

REGISTRANT *NAME AND ADDRESS*

* 000551 BAIRD & MCGUIRE INC
SOUTH STREET
HOLBROOK, MA 02343

********** PRODUCT NAME **********

**00097* PENTA TOX DP

**00102* PENTASOTE 5% SOLUTION OF PENTACHLOROPHENOL

**00103* PENTASOTE CONCENTRATE

**00104* PENTA TOX DP CONCENTRATE

**00151* PCP 10% EMULSIFIABLE CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 000557 SWIFT AGRICULTURAL CHEMICAL
CORP
111 WEST JACKSON BOULEVARD
CHICAGO, IL 60604

********** PRODUCT NAME **********

**01702* VIGORO WEED & GRASS KILLER

*** PRODUCT SEARCH LISTING *** PAGE 17

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000572 ROCKLAND CHEMICAL CO. INC.
P O BOX 809
CALDWELL, NJ 07006

********** PRODUCT NAME **********

**00092* ROCKLAND "PENTA PENN'

**00100* ROCKLAND PENTA CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 000602 RALSTON PURINA COMPANY
CHECKERBOARD SQUARE
ST LOUIS MO 63188

********** PRODUCT NAME **********

**00040* PURINA INSECT OIL CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 000654 FEDERAL CHEMICAL COMPANY INC
SUITE K 1, ATKINSON SQUARE
5820 WEST 85TH ST. SUITE 101
INDIANAPOLIS, IN 46278

********** PRODUCT NAME **********

**00080* ARAB WATER REPELLENT PAINTABLE READY TO USE. WCCI PRESERVATIVE CLEA

**00083* ARAB STAIN 1 COAT DOES 3 JOBS

**** PRODUCT SEARCH LISTING **** PAGE 18

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000713 ATLAS MANUFACTURING COMPANY
PO BOX 4350
DALLAS TX 75208

********** PRODUCT NAME **********

**00007* ATLAS 602 VEGOCIL

**00010* ATLAS 603 DEC VEG

****

REGISTRANT *NAME AND ADDRESS*

* 00769 WOOLFOLK CHEMICAL WORKS INC
PO BOX 938
FT VALLEY GA 31030

********** PRODUCT NAME **********

**00107* ANTIROT 10X CONCENTRATE

**00435* SECURITY 7% PENTA HERBICIDE FOR NON CROPLAND USE ONLY

****

REGISTRANT *NAME AND ADDRESS*

* 00882 LILLY CEAS H COMPANY MILLER RD DIV
7737 N.E KILLINGSWORTH
PORTLAND, OR 97218

********** PRODUCT NAME **********

**00062* MILLER'S PENTA CONCENTRATE 1 TO 10 (A PENTACHLOROPHENOL WOOD PRESER

**** PRODUCT SEARCH LISTING **** PAGE 19

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000891 HERCULES INC AGRICULTURAL CHEMICAL
SYNTHETIC DEPT
WILMINGTON DE 19899
WILMINGTON DE 19899

********** PRODUCT NAME **********

**00128* HERCULES MICROBIOLOGICAL COMPOUND MB 1 8

****

REGISTRANT *NAME AND ADDRESS*

* 000892 PIONEER MFG COMPANY
3753 E 87TH ST
CLEVELAND OH 44104

********** PRODUCT NAME **********

**00030* K O WEED KILLER WITH DRIFT CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 000912 FARMERS UNION CENTRAL EXCHANGE INC
POST OFFICE BOX 'G'
ST PAUL MN 55164

********** PRODUCT NAME **********

**00088* PACIFIC COOPERATIVES WOOD PRESERVATIVE

**** PRODUCT SEARCH LISTING **** PAGE 2

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 00962 LOS ANGELES CHEMICAL COMPANY
4545 ARDINE ST
SOUTH GATE CA 90280

********** PRODUCT NAME **********

**00232* LACCO PENTA CONCENTRATE

**00343* LACCO PENTA WEED KILLER

**00361* LACCO PENTA READY

****

REGISTRANT *NAME AND ADDRESS*

* 001020 OAKITE PRODUCTS INC
50 VALLEY RD
BERKELEY HEIGHTS NJ 07922

********** PRODUCT NAME **********

**00006* OAKITE 55M

****

REGISTRANT *NAME AND ADDRESS*

* 001022 CHAPMAN CHEMICAL COMPANY
BOX 9158
MEMPHIS TN 38109

********** PRODUCT NAME **********

**00004* CHAPMAN PENTA GENERAL WOOD KILLER

**00008* PERMATOX 10 5

**00015* CHAPMAN PENTA PRESERVATIVE CONCENTRATE 1 TO 10

*** PRODUCT SEARCH LISTING **** PAGE 21

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001022

**00016* CHAPMAN PENTA READY TO USE WOOD PRESERVATIVE & SOIL POISON

**00046* POL NU PENTA PRESERVATIVE GREASE

**00068* CHAPMAN PENTA WR CONCENTRATE 1 TO 5 WOOD PRESERVATIVE

**00120* PENTA PLUS 40

**00218* TIMPREG PAK

**00230* PERMATOX 10 S PLUS BAZIDE

**00240* POL NU PAK

**00256* TIMPREG

**00356* PENTA WR READY TO USE

**00368* TIMPREG E POL NU TYPE WOOD PRESERVATIVE GREASE

**00379* TIMPREG E SPECIAL

**00393* TIMPREG E PAK

**00408* CHAPMAN POL NU 15 15 PENTA PRESERVATIVE GREASE WITH CREOSOTE ADDED

**00438* PERMATOX 7 (PRILLED) PENTACHLOROPHENOL, TECHNICAL

**00446* CHAPMAN SURFACE SEALER WOOD PRESERV READY TO USE MIL S 13518B TYPE

**00447* CHAPMAN PERMATOX 7 (OILED)

**00464* CHAPMAN WEED A WAY

**00469* POL KAP GREASE

**00472* WEED A WAY CONCENTRATE

**00474* NAPCLOR G

**00475* BUCHANAN SPECIAL BLEND STAIN CONTROL

**00477* AMBRITE T

**00479* P 110 G

**00480* P 160

**** PRODUCT SEARCH LISTING **** PAGE 22

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001022

**00481* P 180

**00482* PQ 10

**00496* POL NU 20 20

****

REGISTRANT *NAME AND ADDRESS*

* 001043 VESTAL LABORATORIES DIV OF CHEMED CORP
4963 MANCHESTER AVE
ST LOUIS MO 63110

********** PRODUCT NAME **********

**00015* ANTI BACTERIAL VESCO LITE

**00040* TY LON 27 BACTERIOSTAT & ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 001066 CRE O TOX CHEMICAL PRODUCTS COMPANY
BOX 12598
MEMPHIS TN 38112

********** PRODUCT NAME **********

**00004* CRE O TOX A10 WOOD PRESERVING CONCENTRATE

*** PRODUCT SEARCH LISTING **** PAGE 23

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001127 BOURBON CO
BOX 466
LEXINGTON KY 40511

********** PRODUCT NAME **********

**00019* BOURBON WOOD PRESERVATIVE READY TO USE

****

REGISTRANT *NAME AND ADDRESS*

* 001187 VIRGINIA CHEM INC
3340 W NORFOLK RD
PORTSMOUTH VA 23703

********** PRODUCT NAME **********

**00066* VIRGINIA ALGAE CIDE NO. 2

**00085* VIRCHEM 503 MICROORGANISM CONTROL CHEMICAL

****

REGISTRANT *NAME AND ADDRESS*

* 001203 ROBERTS CONSOLIDATED INDUSTRIES, INC.
SUBSIDARY OF CHAMPION INTERNATIONAL CORP.
2305 SUPERIOR AVENUE
KALAMAZOO, MI 49003

********** PRODUCT NAME **********

**00019* FOREMOST 4950 CONCENTRATE

**00050* FOREMOST 4955 ES WEED ZAPPER

**** PRODUCT SEARCH LISTING **** PAGE 24

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001221 BARTLETT CHEMICALS INC
BOX 50730
NEW ORLEANS LA 70125

********** PRODUCT NAME **********

**00024* BARTLETT LIONATOX WOOD PRESERVATIVE PENTACHLOROPHENOL BASE

****

REGISTRANT *NAME AND ADDRESS*

* 001266 MALTER INTERNATIONAL CORPORATION
PO BOX 6099
NEW ORLEANS LA 70114

********** PRODUCT NAME **********

**00052* WEED GC 20 2 1

****

REGISTRANT *NAME AND ADDRESS*

* 001285 CHEMISEAL COMPANY
353 NO WESTERN AVE
CHICAGO, IL 60612

********** PRODUCT NAME **********

**00005* CHEMISEAL NO.100 TOXIC WOOD PRESERVATIVE & WATER REPELLENT

*** PRODUCT SEARCH LISTING **** PAGE 25

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001307 MAGNOLIA CHEMICAL CO INC
2646 RODNEY LANE
DALIAS TX 75220

********** PRODUCT NAME **********

**00120* MAGNOLIA SUPER KILL

****

REGISTRANT *NAME AND ADDRESS*

* 001353 VACCINOL CHEM CO INC
C/O MORTON PHARMACEUTICALS
1625 NORTH HIGHLAND
MEMPHIS TN 38108

********** PRODUCT NAME **********

**00001* VACCINOL 'PENTA" PRESERVATIVE READY TO USE WOOD PRESERVATIVE

**00002* VACCINOL 'PENTA' PRESERVATIVE 1 TO 10 CONC WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 001386 UNIVERSAL COOPERATIVES INC
3001 METRO DR. SUITE 500
MINNEAPOLIS MN 55420

********** PRODUCT NAME **********

**00030* PENTA CONCENTRATE

**00117* UNICO WOOD SAVER

**00524* UNICO KLEEN WALK WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 26

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001409 ROBERTS CONSOLIDATED INDS INC
SUBS. CHAMPION INTERN. CORP.
2305 SUPERIOR AVE
KALAMAZOO, MI 49003

********** PRODUCT NAME **********

**00002* WOODLIFE

**00007* U.S. PLYWOOD PENTACHLOROPHENOL CONCENTRATE CLEAR

**00010* U.S. PLYWOOD PENTACHLOROPHENOL READY TO USE

**00012* WOODLIFE CLEAR CONCENTRATE

**00014* WOODLIFE,, CLEAR READY TO USE

**00015* U. S. PLYWOOD WOODLIFE CLEAR W/PENTA. CONC. CLEAR 4 1

**00021* WRP 146 WATER REPELLENT PRESERVATIVE FOR WOOD READY TO USE CLEAR

**00026* LIFEPRIME REDWOOD CONCENTRATE

**00027* WOODLIFE READY TO USE, BLACK

**00028* LIFEPRIME REDWOOD READY TO USE

**00030* WOODLIFE MILLTREAT TYPE E

**00034* WOODLIFE MILLTREAT TYPE EC

**00038* WOODLIFE EC WATER REPELLENT PRESERVATIVE FOR WOOD

**00047* WOODLIFE MILLTREAT TYPE FD

**00048* WOODLIFE MILLTREAT TYPE E EC CONCENTRATE

**00049* WOODLIFE MILLTREAT TYPE E CONCENTRATE

*** PRODUCT SEARCH LISTING *** PAGE 27

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001421 DETTELBACH CHEMICALS CORP
ATTN WARD A R 4181 PEACHTREE RD NE
ATLANTA GA 30319

********** PRODUCT NAME **********

**00053* PENTA FIVE

**00160* GLOBE SOIL STERILANT

**00161* KEM K O 6 WEED KILLER

**00180* STERI KILL SOIL STERILANT

****

REGISTRANT *NAME AND ADDRESS*

* 001439 BLUE SPRUCE COMPANY
1390 VALLEY ROAD
STIRLING, NJ 07980

********** PRODUCT NAME **********

**00024* CHEM TOL 40% SOLUTION W/W (A 10 TO 1 PONTA CONCENTRATE)

**00054* CHEM TOL 5% SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 001476 GILBREATH CHEMICAL COMPANY
701 16TH ST
SAN FRANCISCO CA 94107

********** PRODUCT NAME **********

**00006* KENITE '9'

**** PRODUCT SEARCH LISTING **** PAGE 28

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001476

**00011* KENITE '101' A 10 1 CONCENTRATED WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 001553 MOMAR INC
PO BOX 19567 STATION N
ATLANTA GA 30325

********** PRODUCT NAME **********

**00070* MOMAR NO MOW WEED AND GRASS KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001616 WARREN DOUGLAS CHEMICAL COMPANY INC
3002 F ST
OMAHA NE 68107

********** PRODUCT NAME **********

00005* WARLASCO*5% PENTACHLOROPHENOL WOOD PRESERVATIVE

**00068* WARLASCO PENTACHLOROPHENOL 10 TO 1 CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 001624 US BORAX & CHEMICAL CORP
3075 WILSHIRE BLVD
LOS ANGELES, CA. 60010

********** PRODUCT NAME **********

**00049* PENTAMUL WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 29

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001624

**00080* PENTAMUL HD WEED KILLER

**00082* PENTAMUL*LHD WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001645 KNOX CHEMICAL CO
7625 PAGE BLVD
ST. LOUIS, MO. 63133

********** PRODUCT NAME **********

**00023* KNOX GENERAL WEED KILLER OIL SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 001678 DYAIL PRODUCTS
35E. BLECKE AVE
ADDISON IL 60101

********** PRODUCT NAME **********

**00002* NO 125 WT5 TOXIC CERASEAL

****

REGISTRANT *NAME AND ADDRESS*

* 001685 STATE CHEMICAL MFG CO THE
3100 HAMILTON AVE
CLEVELAND, OHIO 44114

********** PRODUCT NAME **********

**00043* FORMULA 190 NON SELECTIVE WEEI AND BRUSH KILLER

**** PRODUCT SEARCH LISTING **** PAGE 3

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001685

**00059* FORMULA 267 PARCH NON SELECTIVE HERBICIDE

**00073* FORMULA 267 B PARCH NON SELECTIVE WEED KILLER

**00074* STATEBRAND FORMULA 267 A PARCH NON SELECTIVE HERBICIDE

**00076* FORMULA 190 A NON SELECTIVE WEED AND BRUSH KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001706 NALCO CHEMICALS COMPANY
REGULATORY AFFAIRS CONSULTANT
2901 BUTTERFIELD ROAD
OAK BROOK, IL 60521

********** PRODUCT NAME **********

**00006* NALCO 21 5

**00007* NALCO NO. 21 M

**00015* NALCO H 170 HERBICIDE

**00027* NALCO 201

**00037* NALKIL WEED KILLER 250 LIQUID

**00038* NALKIL WEED KILLER 400 LIQUID

**00054* NALCO 401 MICROORGANISM CONTROL CHEMICAL

**00057* NALCO 424 MICROORGANISM CONTROL CHEMICAL

**00061* NALCO HERBICIDE H 178D

**00079* NALKIL WEED KILLER 250 CONCENTRATE

**00080* NALKIL WEED KILLER 400 CONCENTRATE

**00082* NALKIL WEED KILLER 251 CONCENTRATE

**00086* NALKIL WEED KILLER 401 CONCENTRATE

**00089* NALKIL 2701 LIQUID WITH ENDRIFT WEED KILLER

*** PRODUCT SEARCH LISTING **** PAGE 31

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001706

**00094* NALKIL 2703 LIQUID WITH ENDRIFT WEED KILLER

**00096* NALKIL 2702 LIQUID WITH ENDRIFT WEED KILLER

**00097* NALCO 428 BALL BRIQUETTE MICROORGANISM CONTROL CHEMICAL

**00104* NALKIL 2727 LIQUID WITH ENDRIFT WEED KILLER

**00105* NALKIL 2728 LIQUID WITH ENDRIFT WEED KILLER

**00106* NALKIL 400 CONCENTRATE WITH ENDRIFT WEED KILLER

**00107* NALKIL 2726 LIQUID WITH ENDRIFT WEED KILLER

**00119* NAKIL 250 CONC W/ENDRIFT WEED KILLER

**00120* NALKIL 251 CONC W/ENDRIFT WEED KILLER

**00121* NALKIL 401 CONC W/ENDRIFT WEED KILLER

**00127* NALKIL 2703 WITH ENDRIFT SPRAY WEED KILLER

**00130* NALKIL 600 LIQUID WITH ENDRIFT WEED KILLER

**00131* NALKIL 250 LIQUID WITH ENDRIFT WEED KILLER

**00132* NALKIL 251 LIQUID WITH ENDRIFT WEED KILLER

**00133* NALKIL 601 LIQUID WITH ENDRIFT WEED KILLER

**00134* NALKIL 400 LIQUID WITH ENDRIFT WEED KILLER

**00135* NALKIL 401 LIQUID ENDRIFT WEED KILLER

**00136* NALCO H 170 E HERBICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 001730 AMERICAN CYANAMID COMPANY CONSUMER PRODUCT DIV
ATT MEDICAL DIRECTOR 859 BERDAN AVE
WAYNE NJ 07470

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 32

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 001730

**00014* CYTOX 431 INDUSTRIAL BIOCIDE

****

REGISTRANT *NAME AND ADDRESS*

* 001748 YORK CHEMICAL COMPANY
195 ATLANTIC AVE
GARDEN CITY PARK NY 11044

********** PRODUCT NAME **********

**00092* CERTOX P 40 WOOD PRESERVER

**00094* CERTOX P 5 WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 001757 DREW CHEMICAL CORPORATION
ONE DREW CHEMICAL PLAZA
BOONTON, NEW JERSEY 07005

********** PRODUCT NAME **********

**00009* BIOSPERSE

**00025* BIOCIDE 207

**00042* DREWSPERSE 780 (FOR INDUSTRIAL USE ONLY)

*** PRODUCT SEARCH LISTING *** PAGE 33

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001762 ASSCC CHEMISTS INC
4401 SE JOHNSON CK BLVD
PORTLAND OR 97206

********** PRODUCT NAME **********

**00004* OREGON BRAND FUNGICIDE K 72

****

REGISTRANT *NAME AND ADDRESS*

* 001769 NATIONAL CHEMSEARCH DIV
USACHEM INC
2727 CHEMSEARCH BLVD
IRVING TX 75060

********** PRODUCT NAME **********

**00112* CHEM AQUA 400

**00115* WT 11 WATER TREATMENT

**00180* NATIONAL CHEMSEARCH FENOCIL WEED KILLER KILLS ALL VEGETATION

**00186* NATIONAL CHEMSEARCH CHEM AQUA 800 A COOLING TCWER ALGAECIDE

**00188* NATIONAL CHEMSEARCH ACTI CIL WEED KILLER

**00196* NATIONAL CHEMSEARCH PENTA CIL LOW DRIFT WEED KILLER

**00217* NATIONAL CHEMSEARCH BRIM FAST ACTING RESIDUAL WEED AND GRASS KILLER

**00221* NATIONAL CHEMSEARCH FS 90 WEED KILLER KILLS ALL VEGETATION

**00249* NATIONAL CHEMSEARCH HK 7 WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 34

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001783 TRIC CHEMICAL WORKS INC
341 347 SCHOLES ST
BROCKLYN NY 11200

********** PRODUCT NAME **********

**00058* TRIC WOOD PRESERVATIVE, WATER REPELLENT (PENTACHLOROPHENOL)

****

REGISTRANT *NAME AND ADDRESS*

* 001786 USS CHEMICALS DIV US STEEL CORP
600 GRANT ST (RM 2888)
PITTSBURGH, PA 15230

********** PRODUCT NAME **********

**00007* USS PENTACHLORPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001842 TRIANGLE CHEMICAL COMPANY
BOX 4528
MACON GA 31208

********** PRODUCT NAME **********

**00033* TRIANGLE 5% PENTACHLOROPHENOL WOOD PRESERVATIVE

**00034* TRIANGLE PENTACHLOROPHENOL 10:1 CONC WOOD PRESERV

*** PRODUCT SEARCH LISTING *** PAGE 35

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001864 CENTRAL PETROLEUM COMPANY
548 STANDARD BLDG
CLEVELAND OH 44113

********** PRODUCT NAME **********

**00007* COLUMBIA PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 001898 CROWN TAR & CHEMICAL WORKS INC
900 WEWATTA ST
DENVER CO 80200

********** PRODUCT NAME **********

**00003* PENTACHLOROPHENOL 5% SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 001926 NAVY BRAND MFG COMPANY
5111 SW AVE
ST LOUIS MO 63110

********** PRODUCT NAME **********

**00039* QUICK VEGETATION KILLER & SOIL STERILANT

**** PRODUCT SEARCH LISTING **** PAGE 36

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 001927 TERMINIX DIV OF COCK INC INC
PO BOX 16902
MEMPHIS TN 38116

********** PRODUCT NAME **********

**00058* NO VEX WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 001941 ELCO MANUFACTURING COMPANY
111 3RD ST SHARPSBURG
PITTSBURGH PA 15215

********** PRODUCT NAME **********

**00070* ELCO LUMBER LAST #10

****

REGISTRANT *NAME AND ADDRESS*

* 002155 SCHNEIDI INC
1429 FAIRMONT AVE N W
ATLANTA GA 30318

********** PRODUCT NAME **********

**00051* D WEED O FORMULA #4

**00052* D WEED O FORMULA #5

**00053* D WEED O FORMULA NO 6 NON SELECTIVE WEED KILLER WITH SURE AIM TO R

**00081* MOLLI BAGS

*** PRODUCT SEARCH LISTING **** PAGE 37

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002169 PATTERSON CHEMICAL COMPANY INC
1400 UNION AVE
KANSAS CITY, MO 64101

********** PRODUCT NAME **********

**00033* PATTERSONS PENTACHLOROPHENOL CONCENTRATE 1 TO 10

**00035* PATTERSON'S PENTACHLOROPHENOL PRESERVATIVE READY TO USE

****

REGISTRANT *NAME AND ADDRESS*

* 002217 PBI GORDON CORPORATION
300 SO 3RD ST
KANSAS CITY KS 66118

********** PRODUCT NAME **********

**00215* PENTA 5.

**00216* PENTA 40

**00369* GRASS N WEED VEGETATION KILLER

**00419* TERMI TOX

**00431* TERMI TOX CONCENTRATE

**00538* PERMA KILL PLUS WEED KILLER

**00552* IMPROVED PERMA KILL NO DRIFT WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 38

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002230 WARSAW CHEMICAL COMPANY INC
P O BOX 658 ARGONNE RD
WARSAW IN 46580

********** PRODUCT NAME **********

**00044* BARE WEED AND GRASS KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 002270 HUGE COMPANY INC
7625 PAGE AVE
ST LOUIS, MO 63133

********** PRODUCT NAME **********

**00170* LIQUID WEED KILLER & SEMI SOIL STERILANT NO. 5

****

REGISTRANT *NAME AND ADDRESS*

* 002279 SOUTHERN PROTECTIVE PROD CO
PO BOX 10915
ATLANTA GA 30310

********** PRODUCT NAME **********

**00007* SOUTHERN CLEAR WOOD PRESERVATIVE

*** PRODUCT SEARCH LISTING *** PAGE 39

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002337 DEMERT & DOUGHERTY INC
AEROPAK DIVISION
333 SKOKIE BLVD
NORTHBROOK, IL 60062

********** PRODUCT NAME **********

**00007* D & D BLOXROT O

**00012* D & D WATER REPELLENT PENTA

**00034* D & D CEDARIZER

**00035* D & D PENTA PRIMER AND SEALER

**00040* D & D CONCENTRATE 1 10 BLOXROT C PENTA WOOD PRESERVATIVE

**00059* REDWOODIZER

**00063* DEMERT ALL WEATHER REDWOODIZER

****

REGISTRANT *NAME AND ADDRESS*

* 002342 KERR MCGEE CHEMICAL CORP
MGR PKG & LABELING
KERR MCGEE CENTER
OKLAHOMA CITY OK 73102

********** PRODUCT NAME **********

**00368* FASCO LIQUID FLAME (PROD. NO. 873)

**** PRODUCT SEARCH LISTING **** PAGE 40

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002413 M & M CHEMICAL SALES CORP
1073 HANCOCK STREET
QUINCY, MA 02169

********** PRODUCT NAME **********

**00005* REDWOOD PENTA STAIN PIGMENTED REDWOOD FINISH

**00009* PENTA 5 WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 002439 APPERTON CHEMICAL COMPANY
PO BOX 2555 2903 STRICKLAND ST
JACKSONVILLE FL

********** PRODUCT NAME **********

**00002* PENTA CURE

****

REGISTRANT *NAME AND ADDRESS*

* 002568 JOHN BALTIMORE COPPER PAINT CO.
SCM CORPORATION 501 KEY HIGHWAY
BALTIMORE MD 21230

********** PRODUCT NAME **********

**00004* BALTOCOP PENTA CLEAR WOOD PRESERVATIVE

**** PRODUCT SEARCH LISTING **** PAGE 41

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002619 NL INDUSTRIES INC
BOX 512
SAN FRANCISCO CA 94101

********** PRODUCT NAME **********

**00009* DUTCH BOY WOOD PRESERVATIVE 70 90 CLEAR

****

REGISTRANT *NAME AND ADDRESS*

* 002652 MILLER PURCELL COMPANY
P O BOX 245
NEW LENOX IL 60451

********** PRODUCT NAME **********

**00004* MPC CLEARTOX WOOD PRESERVATIVE

**00005* MPC STAINTOX

****

REGISTRANT *NAME AND ADDRESS*

* 002666 CORNELL CHEMICAL & EQUIPMENT CO. INC.
3500 WASHINGTON BLVD.
BALTIMORE MD 21227

********** PRODUCT NAME **********

**00018* CORNELL PENTA GARD 40

**00070* CORNELL PENTA GARD 5

**** PRODUCT SEARCH LISTING **** PAGE 42

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002829 VENTRON CORP
CHEMICAL DIV
CONGRESS ST
BEVERLY, MA 01915

********** PRODUCT NAME **********

**00094* DUROTEX 7665

**00095* DUROTEX 7674

**00101* DUROTEX 7665A

**00106* MYSTOX LEL 4E

**00107* VENTRON

****

REGISTRANT *NAME AND ADDRESS*

* 002831 NAPASCO INTERNATIONAL, INC
PO DRAWER 1057
THIBADAUX, LA 70301

********** PRODUCT NAME **********

**00011* NAPASCO WEED A WAY NON SELECTIVE WEED AND BRUSH KILLER CONCENTRATE

**00012* WEED A WAY NON SELECTIVE WEED & BRUSH KILLER CONCENTRATE

**00013* NAPASCO 9069 WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 43

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002900 SHEPARDS CHEMICAL WORKS
P. O. BOX 473
WILMINGTON, NC 28401

********** PRODUCT NAME **********

**00002* SHEPARD S PENTA WOOD PRESERVATIVE CLEAR

****

REGISTRANT *NAME AND ADDRESS*

* 002935 WILBUR ELLIS CO
P. O. BOX 1286
FRESNO, CA 93715

********** PRODUCT NAME **********

**00133* RED TOP WOOD PRESERVATIVE CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 003008 OSMOSE WOOD PRES COMPANY OF AMERICAN INC
980 ELLICOTT ST
BUFFALO NY 14209

********** PRODUCT NAME **********

**00001* 1 TO 4 CONCENTRATE PENTOX WOOD PRESERVER PENTACHLOROPHENOL SOLUTION

**00004* OSMOSE WOOD PRESERVING COMPOUND OSMOPLASTIC

**00013* OSMOPLASTIC F

**00014* OSMOBAND WOOD PRESERVATIVE BANDAGE

**00015* OSMOPLASTIC B

**** PRODUCT SEARCH LISTING **** PAGE 44

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 003008

**00019* POLE TOPPER FLUID

**00025* PCP 1 10 CONCENTRATE WOOD PRESERVATIVE

**00028* OSMOSE 24 12 WOOD PRESERVATIVE SOLUTION

**00030* PCP 10% BTU SOLUTION

**00037* OSMOPENTA WOOD PRESERVING COMPOUND

****

REGISTRANT *NAME AND ADDRESS*

* 003070 SOUTHWESTERN PETROLEUM COMPANY
PO BOX 789
FT WORTH TX 76101

********** PRODUCT NAME **********

**00003* SWEPCO WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 003073 WOOLSEY MARINE INDUSTRIES INC
100 SAW MILL RD.
DANBURY, CT 06810

********** PRODUCT NAME **********

**00058* DOLFINITE 3905 TX FUNGICIDAL BEDDING COMPOUND

**00059* DOLFINITE 3951 TX FUNGICIDAL DOUBLE PLANKING COMPOUND

*** PRODUCT SEARCH LISTING *** PAGE 45

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003090 SANITIZED INC
605 3RD AVE
NEW YORK NY 10016

********** PRODUCT NAME **********

**00026* SANITIZED SPX FOR TEXTILES

**00027* SANITIZED SPI

**00030* SANITIZED SP FOR FEATHERS

**00034* SANITIZED SPS FOR LATEX IMPREGNATED SISAL

**00038* SANITIZED NLA

**00064* SANITIZED GT FOR MATTRESS TICKING

**00079* DURAFRESH SP

**00125* SANITIZED C F A

**00133* SANITIZED PP

**00136* SANITIZED PWS

**00138* SANITIZED HJO

**00163* SANITIZED BRAND VAN INTERIOR AEROSOL

**00199* SANITIZED BRAND CFB BACTERIOSTAT

**00205* SANITIZED BRAND CFC BACTERIOSTAT

**** PRODUCT SEARCH LISTING **** PAGE 46

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003098 BAXTER J H & COMPANY
1700 S EL CAMINO REAL
SAN MATEO CA 94402

********** PRODUCT NAME **********

**00011* J. H. BAXTER PENTACHLOROPHENOL SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 003142 FOREAN FORD PAINTS INC
1200 MENDELSSOHN AVE
MINNEAPOLIS MN 55427

********** PRODUCT NAME **********

00003* ARM R BRAND * SEAL TOX (PREPARED)

****

REGISTRANT *NAME AND ADDRESS*

* 003215 TEXAS REFINERY CORP
830 850 N MAIN ST
FT WORTH TX 76101

********** PRODUCT NAME **********

**00001* NEVARCT WOOD PRESERVER

*** PRODUCT SEARCH LISTING *** PAGE 47

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003286 FERI STAFFEL CO.
P O BOX 238?
SAN ANTONIO TX 78298

********** PRODUCT NAME **********

**00029* STAFFELS RIDALL

****

REGISTRANT *NAME AND ADDRESS*

* 003339 PARKE HILL CHEMICAL CORP
29 BERTEL AVE
MT VERNON NY 10550

********** PRODUCT NAME **********

**00 16* ALGAE BAN 'X'

**00018* AIR CON TABLETS

****

REGISTRANT *NAME AND ADDRESS*

* 003350 ARKANSAS COMPANY INC
ATTN A A COCK BOX 21
NEWARK NJ 07102

********** PRODUCT NAME **********

**00008* ARKO MOLD PREVENTOR

**** PRODUCT SEARCH LISTING **** PAGE 48

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003486 AMERICAN TAR COMPANY
1700 N NORTHLAKE WAY
SEATTLE WA 98103

********** PRODUCT NAME **********

**00008* ATCO 1405 PENTA WOOD PRESERVATIVE

**00009* 1412 ATCO PENTA PRESERV A SEAL

**00010* ATCO 1410 PENTA TEN TO ONE CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 003509 SAFE WAY FARM PRODUCTS COMPANY
2519 E FIFTH ST
AUSTIN TX 78702

********** PRODUCT NAME **********

**00115* SAFE WAY BRAND 10 1 PENTACHLOROPHENOL WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 003618 INDUSTRIAL COLLOIDS & CHEMICALS INC
PO BOX 1946
KNOXVILLE, TN 37901

********** PRODUCT NAME **********

**00002* INDUSTRIAL BR PETNACHLOROPHENOL SOL WOOD PRES 12 1/2 1 CONCENT

**** PRODUCT SEARCH LISTING **** PAGE 49

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003658 THE DOLPHIN PAINT & CHEMICAL COMPANY
922 LOCUST STREET
TOLEDO OH 43613

********** PRODUCT NAME **********

**00019* DOLFINITE 1481TX COMPOUND THINNER (FOR FUNGICIDAL COMPOUNDS)

**00026* DOLFINITE 3431 TX FUNG DOUBLE PLANKING COMP(MIL S 19653A TYPE 11,

**00027* DOLFINITE 3435 TX FUNG BED COMP MIL SPEC NO MIL S 19653A TYPE I

****

REGISTRANT *NAME AND ADDRESS*

* 003682 SMITH E W CHEM COMPANY
15020 E PROCTOR AVE
CITY OF INDUSTRY CA 91746

********** PRODUCT NAME **********

**00001* EMS COOLING TOWER& CONDENSER TREATMENT #315

**00019* EMS ALGAECIDE 316

**00020* EMS COOLING TOWER & CONDENSER TREATMENT #313 (CONCENTRATE)

**00024* EMS ALGAECIDE PELLETS 'S'

**00026* EMS COOLING TOWER & CONDENSER TREATMENT #320 B CONCENTRATE

**00029* EMS ALGAECIDE 319

**00032* EMS ALGAECIDE 316

**** PRODUCT SEARCH LISTING **** PAGE 50

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003809 SOUTHWEST GREASE OIL CO INC
KANSAS CITY DIV
BOX 1974 1400 SOUTH HARRISON
CLATHE, KS 66061

********** PRODUCT NAME **********

**00001* NO. 67011 PENTA WOOD PRESERVATIVE

**00007* PENTA CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 003837 LUBAR COMPANY
1708 CAMPBELL
KANSAS CITY MO 64108

********** PRODUCT NAME **********

**00024* BROMA WEED KILLER

**00028* 900 WEED KILLER

**00036* PRIME HERBICIDE

**00040* 501 L

****

REGISTRANT *NAME AND ADDRESS*

* 003862 ABC COMPOUNDING COMPANY
BOX 932
ATLANTA GA 30301

********** PRODUCT NAME **********

**00012* 10 TO 1 PENTA CHLOROPHENOL

**** PRODUCT SEARCH LISTING **** PAGE 51

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 003862

**00013* PENTA CHLOROPHENOL SOLUTION

**00026* WEED KILLER CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 003876 BETZ LABORATORIES, INC
4636 SOMERTON ROAD
TREVOSE, PA 19047

********** PRODUCT NAME **********

**00005* BETZ ALGICIDE WX P

**00012* BETZ ALGICIDE TX P

**00015* BETZ SLIME TROL SLIME CONTROL AGENT

**00026* BETZ SLIMICIDE FORMULA 9 (SLIME CONTROL AGENT)

**00027* BETZ SLIME TROL RX 17

**00030* BETZ FUNGICIDE F 14

**00041* BETZ SLIME TROL RX 26

**00060* BETZ SLIME TROL DP 324

**00065* BETZ SLIMICIDE B 7P (PELLETS)

**00066* BETZ SLIME TROL DP 339

**00067* BETZ FUNGICIDE FUNGUS CONTROL AGENT DE 316

**00079* BETZ FUNGICIDE F 16 FUNGUS CONTROL AGENT

**00097* SLIME TROL RX 25

**00105* SLIME TROL DP 578

**00116* BETZ SLIME TROL RX 12A

**** PRODUCT SEARCH LISTING **** PAGE 52

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003931 ANDERSON CHEMICAL COMPANY
1840 WATERVILLE RD PO BOX 4507
MACON GA 31208

********** PRODUCT NAME **********

**00002* ANCO ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 004000 SOUTHERN CHEMICAL PRODUCTS COMPANY
PO BOX 205
MACON GA 31202

********** PRODUCT NAME **********

**00057* PENTA 5 WEED KILLER

**00061* CLEAN KILL SEASON LONG NON SELECTIVE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 004029 OIL SPECIALTIES & REFINING COMPANY
18 BRIDGE ST
BROOKLYN NY 11201

********** PRODUCT NAME **********

**00031* OSR NON SELECTIVE WEED KILLER WITH NO DRIFT

**** PRODUCT SEARCH LISTING **** PAGE 53

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 004091 BARB W M & CO
P O BOX 1879
MEMPHIS, TN 38101

********** PRODUCT NAME **********

**00003* DEEP TREAT PENTA WOOD PRESERV CONC 1 10 GENERAL PURPOSE

**00004* SEAL TREAT PENTA WOOD PRESERVATIVE

**00005* DEEP TREAT PENTA WOOD PRESERVATIVE READY TO USE

****

REGISTRANT *NAME AND ADDRESS*

* 004154 STEBBINS & ROBERTS INC
BOX 791
LITTLE ROCK AR 72203

********** PRODUCT NAME **********

**00001* PENTA TREET GENERAL PURPOSE WOOD PRESERVATIVE

**00002* PENTA CHEX WOOD PRESERVATIVE

**00004* PENTA CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 004243 GINN J R & COMPANY
1401 GRATIOT ST
ST LOUIS MO 63103

********** PRODUCT NAME **********

**00002* GINCO PRODUCTS READY TO APPLY WATER REPELLENT PENTA

**** PRODUCT SEARCH LISTING **** PAGE 54

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 004243

**00003* GINCO NO. 10 PENTA CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 004297 RELIANCE BROOKS INC
3302 E 87TH STREET
CLEVELAND OH 44127

********** PRODUCT NAME **********

**00015* H C TREATMENT NO 475 C FOR SLIME & ALGAE CONTROL

**00016* H C TREAT NO 475 P FOR SLIME & ALGAE CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 004313 CARROLL COMPANY THE
MFG BY THECARROLL CO. 2900 W. KINGSLEY RD.
GARLAND TX 75040

********** PRODUCT NAME **********

**00047* WEED GO WITH DRIFT CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 004389 PACIFIC CHEMICAL DIV PACE NATIONAL CORP
500 7TH AVE S
KIRKLAND WA 98033

********** PRODUCT NAME **********

**00027* MICROCIDE L 36 ALGAECIDE AND BACTERICIDE

*** PRODUCT SEARCH LISTING *** PAGE 55

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 004389

**00034* PACE PENTA

**00069* POLE TOP FLUID

**00072* N 135 ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 004499 FLOYD PINE PROD COMPANY
BOX 25
ANDALUSIA AL 36420

********** PRODUCT NAME **********

**00003* PENTA PINE WOOD PRESERVER

****

REGISTRANT *NAME AND ADDRESS*

* 004643 DEARBORN CHEMICAL DIV WR GRACE & COMPANY
320 GENESSEE ST
LAKE JURICH IL 60047

********** PRODUCT NAME **********

**00005* DEARCIDE 711

**00012* DEARCIDE 712

**00013* A 101 BRIQUETTE ALGAECIDE AND BACTERIOSTAT

*

**** PRODUCT SEARCH LISTING **** PAGE 56

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 004664 MCCLOSKEY VARNISH COMPANY
7600 STATE RD
PHILADELPHIA PA 19136

********** PRODUCT NAME **********

**00006* MCCLOSKEY ROTT FYTER

**00007* MCCLOSKEY ROTT FYTER CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 004715 COLORADO INTERNATIONAL CORP
7800 MARBLE N E
ALBUQUERQUE, N.M. 87110

********** PRODUCT NAME **********

**00099* BEST 4 SERVIS BRAND PENTA 40

**00138* BEST 4 SERVIS BRAND PENTA KIL CONCENTRATE

**00154* BEST 4 SERVIS BRAND PENTA 5

****

REGISTRANT *NAME AND ADDRESS*

* 004887 STEPHENSON CHEM COMPANY INC
BOX 87188
COLLEGE PARK GA 30337

********** PRODUCT NAME **********

**00013* PENTA 10:1,40% PENTACHLORO PHENOL CONCENTRATE

*** PRODUCT SEARCH LISTING *** PAGE 57

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 004931 GOOD LIFE CHEMICALS INC
BOX 687
EFFINGHAM IL 62401

********** PRODUCT NAME **********

**00031* GOOD LIFE PENTA TREET WOOD PRESERVATIVE

**00034* NEW: PENTA CONCENTRATE WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 004990 JET AER CORPORATION
100 SIXTH AVE
PATERSON NJ 07524

********** PRODUCT NAME **********

**00015* HEDDY WEEDS OUT

****

REGISTRANT *NAME AND ADDRESS*

* 005011 CARREL CHEMICAL CORPORATION
BOX 406
WESTFIELD IN 46074

********** PRODUCT NAME **********

**00140* KEM KIL

**** PRODUCT SEARCH LISTING **** PAGE 58

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005135 WATER SERVICES DIV UNIVERSAL OIL PRODUCTS COMPANY
700 SOUTH FLOWER ST
BURBANK CA 91502

********** PRODUCT NAME **********

**00010* M 60 ALGAECIDE

**00012* CHEMICATOR REFILL TUBE A 8

**00018* UOP M 90 ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 005185 BIO LABS INC
627 E COLLEGE AVE PO BOX 1489
DECATUR GA 30031

********** PRODUCT NAME **********

**00032* BIO GUARD 120/140

****

REGISTRANT *NAME AND ADDRESS*

* 005197 KEM MFG CORP
KEM INTERNATIONAL BLDG
TUCKER GA. 30084

********** PRODUCT NAME **********

**00032* NORKEM 60

**00047* NORKEM 600 C

**** PRODUCT SEARCH LISTING **** PAGE 59

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005211 REICHHOLD CHEMICALS INC
2340 TAYLOR WAY
TACOMA WA 98401

********** PRODUCT NAME **********

**00011* RCI 49 162 PENTACHLOROPHENOL (TECHNICAL GRADE)

**00013* RCI CHLOROPHEN 49 405, 5% PENTACHLOROPHENOL SOLUTION

**00017* CHLOROPHEN 49 400

**00020* CHLOROPHEN 49 402

**00021* RCI 49 409 42% TECHNICAL GRADE PENTACHLOROPHENOL CONCENTRATE

**00022* RCI CHLOROPHEN 49 410

**00023* RCI CHLOROPHEN 49 412

**00024* RCI CHLOROPHEN 49 411

**00025* 49 164 SODIUM PENTACHLOROPHENATE 21% AQUEOUS SOLUTION, TECHNICAL GR

**00026* 49 169 2:1 PENTACHLOROPHENOL TETRACHLOROPHENOL (SHOTTED, TECHNICAL)

**00027* RCI 49 167 COMMERCIAL GRADE PENTACHLOROPHENOL (LUMP)

**00028* RCI 49 413 SODIUM TETRA/PENTA CHLOROPHENATE SOLUTION

**00032* RCI 49 165 SODIUM PENTACHLOROPHENATE

****

REGISTRANT *NAME AND ADDRESS*

* 005266 FORESTRY ENTERPRISES INC
CHAPMAN CHEMICAL COMPANY 416 BROOKS RD
MEMPHIS TN 38109

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 60

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 005266

**00009* K BAN PC 15 WOOD PRESERVATIVE GREASE

****

REGISTRANT *NAME AND ADDRESS*

* 005285 MICHLIN CHEM CORP
9045 VINCENT
DETROIT MI 48211

********** PRODUCT NAME **********

**00015* MR THINZIT PENTA WOOD PRESERVATIVE

**00031* MISTER THINZIT PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 005351 FULTON CHEMICAL COMPANY
P O DRAWER 160
SUMTER SC 29150

********** PRODUCT NAME **********

**00006* 41 40 2 PENTA WOOD PRESERVATIVE O

**00007* 41 4000 PENTA WOOD PRESERVATIVE R

**00008* 41 4004 PENTA CONCENTRATE C 10

*** PRODUCT SEARCH LISTING *** PAGE 61

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005382 VULCAN MATERIAL COMPANY CHEMICAL DIV
P O BOX 7689
BIRMINGHAM AL 35223

********** PRODUCT NAME **********

**00016* NEW: FRONTIER GLAZD PENTA

**00036* VULCAN PREMIUM FOUR POUND PENTA (PCP 2) CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 005427 WRIGHT CHEMICAL CORP
1319 W WABANSIA AVE
CHICAGO IL 60622

********** PRODUCT NAME **********

**00014* WRICO ALG

**00022* ALCORIN T

**00025* WRICO DAR

**00026* ALCORIN T Y

**00027* WRICO DAS

**00029* WRICO LFB

**00030* WRICO RLC

**00033* WRICO DBB

**00039* WRICO RTB

**00045* WRICO PTB

**** PRODUCT SEARCH LISTING **** PAGE 62

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005576 REGAL SUPPLY & CHEMICAL COMPANY
BOX 1955
EL PASO TX 79950

********** PRODUCT NAME **********

**00031* ENDWEED

**00032* STRILATE

****

REGISTRANT *NAME AND ADDRESS*

* 005602 HUB STATES CORPORATION
2002 NORTH ILLINOIS STREET
INDIANAPOLIS IN 46202

********** PRODUCT NAME **********

**00025* PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 005640 HANSEN KINNEY COMPANY
BOX 1203
GREAT FALLS MT 59401

********** PRODUCT NAME **********

**00001* PENTA CHLOROPHENOL WOOD PRESERVATIVE READY TO USE

**00002* PENTA CHLOROPHENOL WOOD PRESERVATIVE CONCENTRATE 1 TO 10

**** PRODUCT SEARCH LISTING **** PAGE 63

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005736 DUBCIS RESEARCH LABORATORIES
DUBCIS CHEM DIV CHEMED CORP 3630 E KEMPER ROAD
SHARONVILLE OH 45241

********** PRODUCT NAME **********

**00032* DUIN 24 NON SELECTIVE WEED AND GRASS KILLER

**00033* DUIN 70

**00053* GAX 15 MICROBIOCIDE

****

REGISTRANT *NAME AND ADDRESS*

* 005831 GILSONITE INC
2946 NE COLUMBIA BLVD
PORTLAND OR 97211

********** PRODUCT NAME **********

**00002* BARTEL'S REDY COAT PENTALENE WOOD PRESERVATIVE NO 454

**00003* BARTEL'S REDY COAT COPPER WOOD PRESERVATIVE #456

**00004* BARTEL'S REDY COAT PENTA CONCENTRATE NO. 453

**00008* BARTEL'S REDY COAT PENTA WOOD PRESERVATIVE 455

****

REGISTRANT *NAME AND ADDRESS*

* 005839 BRUDER M A & SON
52ND & GRAYS AVE
PHILA PA 19143

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 64

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 005839

**00002* M A B NO LASTIC WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 006125 BIXON CHEMICAL CORP
50 19 97TH PLACE
CORONA NY 11368

********** PRODUCT NAME **********

**00018* WEED KILLER WITH DRIFT CONTROL

**00019* WEED KILLER B WITH DRIFT CONTROL

**00023* WEED KILLER C WITH DRIFT CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 006173 CONSOLIDATED PAINT & VARNISH CORPORATION
EAST OHIO BUILDING
CLEVELAND OH 44114

********** PRODUCT NAME **********

**00003* GOODYEAR PENTA WOOD PRESERVER

**00004* HIGHEST QUALITY GOODYEAR BLACK WOOD PRESERVER

*** PRODUCT SEARCH LISTING *** PAGE 65

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006190 SANICO
13143 SATICOY ST
N HOLLYWOOD CA 91605

********** PRODUCT NAME **********

**00018* IDEAL CONCENTRATED WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 006202 PIONEER BLDG SPECIALTIES
1125 SE DIVISION ST
PORTLAND OR 97202

********** PRODUCT NAME **********

**00002* PIONEER BRAND 5% PENTA WOOD PRESERVING COMPOUND W/WATER REP

**00003* PIONEER BRAND PENTA PRESERVATIVE CONCENTRATE 1TO10

**00005* PIONEER BRAND COPPER NAPHTHENATE

****

REGISTRANT *NAME AND ADDRESS*

* 006314 POLE TREATING CO
BLAIR MILL ROAD
WILLOW GROVE, PA 19090

********** PRODUCT NAME **********

**00001* POLE TOX WOOD PRESERVING COMPOUND

**00002* POLEX WOOD PRESERVING COMPOUND

**** PRODUCT SEARCH LISTING **** PAGE 66

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006354 NANKEE ALUMINUM PAINT CO INC
ENGINEER LANE
FARMINGDALE, NY 11735

********** PRODUCT NAME **********

**00004* NANKEE PENTA TREAT WATER REPELLENT WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 006381 GARMAN COMPANY INC
1252 GROVER ROAD
ST LOUIS MO 63125

********** PRODUCT NAME **********

**00005* VAPCO SLIME X

****

REGISTRANT *NAME AND ADDRESS*

* 006482 TEXAS FARM PRODUCTS COMPANY
PO BOX 9
NACOGDOCHES TX 75961

********** PRODUCT NAME **********

**00004* PENTA CON 1 TO 10 CONCENTRATE WOOD PRESERVATIVE AND SOIL POISON

***** PRODUCT SEARCH LISTING ***** PAGE 67

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006527 HAMELET & HAYES COMPANY
COLONIAL ROAD
SALEM MA 01970

********** PRODUCT NAME **********

**00003* KITO H

**00005* KITO G

**00007* STEROZOL N GERMICIDE

**00008* STEROZOL S GERMICIDE

**00009* STEROZOL T PRESERVATIVE

**00010* STEROZOL U PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 006621 EAGLE CHEMICAL COMPANY
2819 W LAKE ST
CHICAGO IL 60612

********** PRODUCT NAME **********

**00056* MINUS 24 WEED AND BRUSH CONTROL

**00057* WEED KILLER #6

**00058* LIQUID WEED KILLER #1

**** PRODUCT SEARCH LISTING **** PAGE 68

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006651 SONFORD PRODUCTS CORPORATION
PO BOX 5570
JACKSON MS 39208

********** PRODUCT NAME **********

**00001* STA BRITE

**00002* STA RITE 14

**00003* STA RITE WATER REPELLENT PRESERVATIVE FOR WOOD

**00013* PENTA CARE READY TO USE PENTA WOOD PRESERVATIVE MULTI PURPOSE

**00015* SONTOX S

**00017* SP 35

**00020* SP 60

**00028* STA BRITE LIQUID

****

REGISTRANT *NAME AND ADDRESS*

* 006720 SOUTHERN MILL CREEK PRODUCTS COMPANY INC
BOX 1096
TAMPA FL 33601

********** PRODUCT NAME **********

**00232* SMCP WEEDOX G/R 250 NON SELECTIVE HERBICIDE

*** PRODUCT SEARCH LISTING *** PAGE 69

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006732 WEILER INSPEC SERV INV J H
5909 N WASHINGTON ST
DENVER CO 80216

********** PRODUCT NAME **********

**00003* POLE AID NO 3

****

REGISTRANT *NAME AND ADDRESS*

* 006735 TIDE PRODUCTS INC
ATTN MW MARSH BOX 1020
EDINBURG TX 78539

********** PRODUCT NAME **********

**00138* TIDE PENTA A 10 TO 1 CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 006762 STERN CHEM CORP
BOX 5070
MONROE LA 71201

********** PRODUCT NAME **********

**00017* WEEDEX 10 NON SELECTIVE FAST ACTING WEED & BRUSH KILLER

**00027* PRESERVIT A

**00028* PRESERVIT B A READY TO USE WOOD PRESERV & SOIL POISON TERMITES

**** PRODUCT SEARCH LISTING **** PAGE 70

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006811 RESEARCH PRODUCTS COMPANY
2423 MERRELL RD
DALLAS TX 75229

********** PRODUCT NAME **********

**00029* REPCO KILL LIQUID WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 006823 WYCKOFF COMPANY
560 WHITE HENRY STUART BLDG
SEATTLE, WA 98101

********** PRODUCT NAME **********

**00002* PENTACHLOROPHENOL SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 006837 WILMAR INC
BOX 3111
CHARLOTTE NC 28203

********** PRODUCT NAME **********

**00056* WIL KIL

**** PRODUCT SEARCH LISTING **** PAGE 71

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006962 MADISON BIONICS
11250 W ADDISON ST
FRANKLIN PARK IL 60131

********** PRODUCT NAME **********

**00020* SEAR 21 PLUS

**00031* QUELONOL

****

REGISTRANT *NAME AND ADDRESS*

* 006993 GERMAINS INC
4820 E 50TH ST
LOS ANGELES CA 90058

********** PRODUCT NAME **********

**00013* GERMAIN'S NON SELECTIVE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 007109 ST LOUIS PAINT MANUFACTURING COMPANY
2500 OHIO AVE
ST LOUIS MO 63104

********** PRODUCT NAME **********

**00004* CHEKR BRAND 5% WOOD PRESERVATIVE READY TO USE

**** PRODUCT SEARCH LISTING **** PAGE 72

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 007122 AR CHEMICAL CORP
1514 ELEVENTH ST
PORTSMOUTH OH 45662

********** PRODUCT NAME **********

**00073* GUARDIAN PENTACHLOROPHENOL 5

**00100* GUARDIAN PENTACHLOROPHENOL 40% PRODUCT NO. 6540

****

REGISTRANT *NAME AND ADDRESS*

* 007135 SUNNYSIDE PRODUCTS INC
5530 N WOLCOTT AVE
CHICAGO IL 60640

********** PRODUCT NAME **********

**00001* SUNNYSIDE READY TO USE PENTA PRESERVATIVE

**00002* PENTA WOOD PRESERVATIVE (WATER REPELLENT)

****

REGISTRANT *NAME AND ADDRESS*

* 007161 NATIONAL SOLVENT CORP
955 W SMITH RD
MEDINA OH 44256

********** PRODUCT NAME **********

**00008* NASCO WATER REPELLENT AND PENTA WOOD PRESERVATIVE

**** PRODUCT SEARCH LISTING **** PAGE 73

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 007234 FORSHAW CHEMICALS INC
650 STATE STREET PO BOX 6055
CHARLOTTE NC 28208

********** PRODUCT NAME **********

**00001* NO MO STANE

**00007* PENWAR

**00009* LUMBERITE

**00011* PENTACON 40

**00015* NO MO STANE IV

**00016* PENWAR 1 5 WOOD PRESERVATIVE CONCENTRATE

**00018* CREE PEN

**00026* PRE CARE WOOD PRESERVATIVE

**00032* NO MO STANE LIQUID

**00045* FORKIL LIQUID WEED KILLER(CONTAINING DRIFT CONTROL ADDITIVES)

**00057* FORPEN 5

**00060* PENTACON 5

**00061* PENTACON 7

**00062* PENTACON 10

**00072* LUMBERITE RONDETS

**** PRODUCT SEARCH LISTING **** PAGE 74

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 007273 CROWN CHEMICAL INCORPORATED
4995 NORTH MAIN STREET
ROCKFORD IL 61101

********** PRODUCT NAME **********

**00081* BRUSH OFF LIQUID WEED KILLER

**00115* WOODTREAT TC PENETRATING PENTA COATING WITH HEPTACHLOR

**00138* CROWN WOODTREAT GEL PENETRATING PENTA COATING

****

REGISTRANT *NAME AND ADDRESS*

* 007298 BACTOSAN INCORPORATED
605 3RD AVE
NEW YORK NY 10016

********** PRODUCT NAME **********

**00001* DURA FRESH SP FOR FEATHERS

****

REGISTRANT *NAME AND ADDRESS*

* 007299 BRENCO CORPORATION
704 NORTH FIRST STREET
ST LOUIS, MO 63102

********** PRODUCT NAME **********

**00005* BRENCO #540 ALGAECIDE

**00013* BENCO #565 LIQUID ALGAECIDE

**** PRODUCT SEARCH LISTING **** PAGE 75

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 007361 PRESERVATIVE PAINT COMPANY
5400 5410 AIRPORT WAY
SEATTLE WA 98108

********** PRODUCT NAME **********

**00001* PENTA WOOD PRESERVATIVE AND SEALER

****

REGISTRANT *NAME AND ADDRESS*

* 007424 JASCO CHEMICAL CORP
P.O. BOX J 1710 VILLA ST.
MOUNTAIN VIEW CA 94042

********** PRODUCT NAME **********

**00002* JASCO PENTA 5

**00003* JASCO PENTA 40

****

REGISTRANT *NAME AND ADDRESS*

* 007500 GIBSON HOMANS COMPANY
2366 WOODHILL RD
CLEVELAND OH 44106

********** PRODUCT NAME **********

**00001* BLACK JACK 5% PENTA WATER REPELLANT WOOD PRESERVING COMPOUND

**00003* BLACK JACK COPPER NAPTHENATE(W/PENTACHLOROPHENOL) WOOD PRESERV COM

**00007* NORTHERN BRAND COPPER NAPHTHENATE

**00008* NORTHERN PENTA CONCENTRATE 1 TO 10

**** PRODUCT SEARCH LISTING **** PAGE 76

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 007500

**00009* NORTHERN PENTA WOOD PRESERVER

**00012* BLACK JACK MULTI PURPOSE HERBICIDE WEED KILLER

**00014* BLACK JACK X MULTI PURPOSE HERBICIDE WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 007547 WESTERN CHEM. CO.
1345 TANEY
NO KANSAS CITY MO 64116

********** PRODUCT NAME **********

**00002* WESTERN ALGAECIDE DRY

**00005* WESTERN ALGAECIDE C CONCENTRATED LIQUID

**00006* WESTERN ALGAECIDE REGULAR LIQUID

**00007* WESTERN ALGAE & SLIME CONTROL D

**00008* WESTERN ALGAE & SLIME CONTROL R REGULAR LIQUID

****

REGISTRANT *NAME AND ADDRESS*

* 007572 BLUE WHITE CHEMICAL COMPANY
88 WELDON PKWY/PROGRESS WEST
ST LOUIS, MO 63043

********** PRODUCT NAME **********

**00009* BLUE WHITE GROUND STERILANT #2 CONTACT WEED KILLER

```
                    **** PRODUCT SEARCH LISTING ****                    PAGE      77

10/02/78        FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

*************************************************************************************

  *REGISTRANT*        *NAME AND ADDRESS*

  *  007621        CONROE CREOSOTING COMPANY
                   PO BOX 9
                   CONROE TX 77301

    **************** PRODUCT NAME ****************

   **00001*  PENTA WOOD PRESERVATIVE READY TO USE

*************************************************************************************

  *REGISTRANT*        *NAME AND ADDRESS*

  *  007701        CHEMICAL SPECIALTIES INC
                   149 WEST TRIGG AVENUE
                   MEMPHIS TN 38106

    **************** PRODUCT NAME ****************

   **00030*  PENTA WOOD PRESERVATIVE

   **00057*  PENTA GENERAL WEED KILLER

*************************************************************************************

  *REGISTRANT*        *NAME AND ADDRESS*

  *  007714        CARTER MARY INDUSTRIES INC
                   PO BOX 23387
                   TAMPA FL 33622

    **************** PRODUCT NAME ****************

   **00003*  MARY CARTER'S REDWOOD STAIN AND WOOD PRESERVATIVE
```

**** PRODUCT SEARCH LISTING **** PAGE 78

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 007779 HOUGHTON E F & CO
MADISON & VAN BUREN AVENUES
NORRISTOWN PA 19401

********** PRODUCT NAME **********

**00008* SLIMEX NC 15

****

REGISTRANT *NAME AND ADDRESS*

* 007909 ATLANTIC CHEMICAL & EQUIPMENT COMPANY
BOX 19697
ATLANTA GA 30300

********** PRODUCT NAME **********

**00001* ACE ALGAECIDE BRIQUETTES IN AUTOMATIC FEEDER CAN

**00003* ACE ALGAECIDE LIQUID NO. 2

**00004* ACE ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 008047 POLY CHEM INC
P. O. BOX 10026
NEW ORLEANS, LA 70121

********** PRODUCT NAME **********

**00018* WEED KILLER NO. 46 FOR GENERAL WEED CONTROL

*** PRODUCT SEARCH LISTING **** PAGE 79

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 008123 MILLER FRANK & SONS INC
13831 SO EMERALD AVE
CHICAGO IL 60627

********** PRODUCT NAME **********

**00023* NON SELECTIVE VEGETATION KILLER (LVK 33)

**00025* LIQUID VEGETATION KILLER

**00028* COMPLETE VEGETATION KILLER

**00029* WEED KILLER AND SEMI SOIL STERILANT

**00069* NON SELECTIVE HERBICIDE

**00070* TOTAL VEGETATION KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 008133 CHAMPION CHEMICAL INC
RES & DEV
BOX 45509
HOUSTON, TX 77045

********** PRODUCT NAME **********

**00018* BACTRON KM 4 MICROBIOCIDE

****

REGISTRANT *NAME AND ADDRESS*

* 008145 BURGESS FOBES PAINT CO
BOX 106
PORTLAND ME 04104

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 80

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 008145

**00001* CU PRE SERV 4192 CLEAR PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 008222 LING FUANG INDUSTRIES, INC
LING FUANG, IND
P.O. BOX 1207
GARDNERVILLE, NV 89410

********** PRODUCT NAME **********

**00020* TOXIC GRASS AND WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 008300 ATLAS CHEMICAL CO
4801 NW 77TH AVE
MIAMI FL 33144

********** PRODUCT NAME **********

**00003* NO. 105 WOOD CARE CONCENTRATE WATER REPELLENT WOOD PRESERVATIVE

**00004* NO 103 WOOD CARE CONCENTRATE WATER REPELLENT WOOD PRESERVATIVE

**00005* WOODCARE WOOD PRESERVATIVE WITH PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 008486 EASTERN CHEMICAL SERVICE
368 BROAD ST
NEW LONDON CT 06320

********** PRODUCT NAME **********

**** PRODUCT SEARCH LISTING **** PAGE 81

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 008486

**00002* EASTERN CHEMICAL SERVICE WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 008513 HARRIS PAINT COMPANY
BOX 1381
TAMPA FL 33601

********** PRODUCT NAME **********

**00007* MIL DU WASH

****

REGISTRANT *NAME AND ADDRESS*

* 008540 GARRATT CALLAHAN COMPANY
111 ROLLINS RD
MILLBRAE CA 94031

********** PRODUCT NAME **********

**00001* GARRATT CALLAHAN FORMULA NO. 30

****

REGISTRANT *NAME AND ADDRESS*

* 008550 PRO SPECIALTIES, INC
7754 W. HARWOOD AVENUE
MILWAUKEE, WI 53213

********** PRODUCT NAME **********

**00010* DEL KILL WEED KILLER 400 LIQUID

**00017* RAPID KILL LIQUID WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 82

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 008550

**00032* LEVEL IT

****

REGISTRANT *NAME AND ADDRESS*

* C08590 AGWAY INC
FERTILIZER CHEMICAL DIVISION
BOX 4933
SYRACUSE NY 13221

********** PRODUCT NAME **********

**00029* AGWAY PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 008591 MOGUL CORPORATION THE
P C BOX 20
CHAGRIN FALLS, OH 44022

********** PRODUCT NAME **********

**00004* MOGUL AG 420

**00005* MOGUL A 423

**00006* MOGUL A 421

**00007* MOGUL A 422

***** PRODUCT SEARCH LISTING ***** PAGE 83

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 008637 MITCO INC
1601 STELL AVE SW
GRAND RAPIDS MI 49502

********** PRODUCT NAME **********

**000004* MITCO CC 30 ALGICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 008643 CROSBY FOREST PRODUCTS COMPANY
BOX 520
PICAYUNE MS 39466

********** PRODUCT NAME **********

**000003* PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 008689 AMBROID COMPANY INC
612 MONTELLO ST
BROCKTON MA 02401

********** PRODUCT NAME **********

**000002* PENTA SAN "20"

**** PRODUCT SEARCH LISTING **** PAGE 84

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 008728 PRECISION CHEMICALS INC
PO BOX 19793 STATION N
ATLANTA GA 30325

********** PRODUCT NAME **********

**00001* ALGAECIDE BRIQUETTES IN AUTOMATIC FEEDER CAN

**00002* ALGAECIDE BRIQUETTES KILLS ALGAE

**00003* ALGAECIDE LIQUID 205

**00004* PRECISION ALGAECIDE LIQUID CL-208

****

REGISTRANT *NAME AND ADDRESS*

* 008790 REPUBLIC POWDERED METALS INC
2628 PEARL RD
MEDINA OH 44256

********** PRODUCT NAME **********

**00001* STOP ROT WOOD PRESERVATIVE & SOIL POISON FOR TERMITES

****

REGISTRANT *NAME AND ADDRESS*

* 008815 EMPIRE VARNISH COMPANY
9808 MEECH AVE
CLEVELAND OH 44105

********** PRODUCT NAME **********

**00001* CLEAR WOOD PRESERVER

**** PRODUCT SEARCH LISTING **** PAGE 85

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 008823 HABCO INC
10501 WAYZATA BLVD. SUITE 101
HOPKINS, MN 55343

********** PRODUCT NAME **********

**00021* HABCO 444 (LIQUID WEED KILLER)

**00023* HABCO 444 CONCENTRATE LIQUID WEED KILLER

**00035* HABCO 233 LIQUID WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 009027 GILMAN PAINT & VARNISH COMPANY
BOX 1257
CHATTANOOGA TN 37401

********** PRODUCT NAME **********

**00001* GILMAN 10 1 PENTOX WOOD PRESERVATIVE

**00002* GILMAN PENTOX WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 009205 AGRO CHEM INC
7855 D GROSSE POINT ROAD
SKOKIE, IL 60076

********** PRODUCT NAME **********

**00012* KILL & PREVENT #301 NON SELECTIVE VEGETATION KILLER & SEMI SOIL STE

**00022* AGRO CHEM BARE EARTH NO. 303

**** PRODUCT SEARCH LISTING **** PAGE 86

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 009285

**00023* AGRO CHEM BARE EARTH CONCENTRATE NO 3 4

****

REGISTRANT *NAME AND ADDRESS*

* 009285 UNITED LABORATORIES INC
155 S RT 53
ADDISON IL 60101

********** PRODUCT NAME **********

**00015* UL 85 WEED AND BRUSH KILLER

**00022* UL 285 WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 009284 WATERLOX CHEMICAL & COATING CORP
9808 MEECH AVE
CLEVELAND OH 44105

********** PRODUCT NAME **********

**00001* WATERLOX RED WOOD PRESERVER

**00002* WATERLOX WOOD STAIN PRESERVER FOR ALL WOOD SURFACES CHESTNUT BROWN

**00003* WATERLOX WOOD STAIN PRESERVER FOR ALL WOOD SURFACES TEAKWOOD GRA.

**00004* WATERLOX WOOD STAIN PRESERVER FOR ALL WOOD SURFACES PERSIMMON BROWN

**00005* WATERLOX WOOD STAIN PRESERVER FOR ALL WOOD SURFACES WILLOW GREEN

**00006* WATERLOX WOOD STAIN PRESERVER FOR ALL WOOD SURFACES STAIN WHITE

**** PRODUCT SEARCH LISTING **** PAGE 87

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009313 ROSE CHEMICAL PROD INC
PO BOX 23375 545 STIMMEL RD
COLUMBUS OH 43223

********** PRODUCT NAME **********

**00015* VOID

****

REGISTRANT *NAME AND ADDRESS*

* 009319 CUSTOM CHEMICALS, INC
476 HESTER STREET
SAN LEANDRO, CA 94577

********** PRODUCT NAME **********

**00034* CUSTOM WEEDICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 009367 THECOCHEM LABS
BOX 15367
TAMPA FL 33614

********** PRODUCT NAME **********

**00013* SK 368 WEED KILLER READY TO USE

**** PRODUCT SEARCH LISTING **** PAGE 88

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009403 ALKEN MURRAY CORP
111 5TH AVE
NEW YORK NY 10003

********** PRODUCT NAME **********

**00001* ALKEN FUNGUS TREATMENT V 2

**00002* ALKEN FUNGUS TREATMENT #1

****

REGISTRANT *NAME AND ADDRESS*

* 009420 WATER CHEMISTS INC
1275 S BOYLE AVE
LOS ANGELES CA 90023

********** PRODUCT NAME **********

**00002* ALGICIDE 101

****

REGISTRANT *NAME AND ADDRESS*

* 009461 CELANESE COATINGS & SPECIALTIES CO
REGULATORY SERV
P O BOX 99038
JEFFERSONTOWN, KY 40299

********** PRODUCT NAME **********

**00089* DEVOE MARINE SUPER TROPICAL ANTI FOULING SHIP BOTTOM PAINT MD 3835

*** PRODUCT SEARCH LISTING *** PAGE 89

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009519 SAVOGRAN COMPANY
259 LENOX ST
NORWOOD MA 02062

********** PRODUCT NAME **********

**00001* WOODIE PENTA

****

REGISTRANT *NAME AND ADDRESS*

* 009573 HARBOR CHEMICAL & ENGR CORP
100 WEST MONROE ST SUITE 706
CHICAGO, IL 60603

********** PRODUCT NAME **********

**00001* HARBCO CT 85 POWDER

**00002* HARBCO CT 86 BEADS

****

REGISTRANT *NAME AND ADDRESS*

* 009578 CWC INDUSTRIES, INC
2750 GRAND AVE
CLEVELAND OH 44104

********** PRODUCT NAME **********

**00001* ALGAECIDE FORMULA CW 70

**** PRODUCT SEARCH LISTING **** PAGE 9^

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009594 INTERCONTINENTAL CHEMICAL CORP
BOX 15318
SACRAMENTO CA 95813

********** PRODUCT NAME **********

**00010* ICC W 66

**00011* ICC W 68

****

REGISTRANT *NAME AND ADDRESS*

* 009639 WILLARD PRODUCTS
BOX 1249
REDWOOD CITY CA 94064

********** PRODUCT NAME **********

**00006* PENTA TREAT 10 TO 1 CONCENTRATE

**00007* PENTA TOX

**00014* PENTA TREAT 300

****

REGISTRANT *NAME AND ADDRESS*

* 009678 SCHWARTZ CHEMICAL COMPANY INC
1041 SEMINOLE BLVD
CASSELBERRY FL 32707

********** PRODUCT NAME **********

**00002* STA KLEEN X RAY FUNGISTATIC SOLUTION

*** PRODUCT SEARCH LISTING *** PAGE 91

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009702 ALLSTATE
BOX 3040
EUCLID OH 44117

********** PRODUCT NAME **********

**00001* ALLSTATE AC PH

****

REGISTRANT *NAME AND ADDRESS*

* 009708 PABCO PAINT CORP
5850 SHELLMOUNT ST
EMERYVILLE CA 94608

********** PRODUCT NAME **********

**00002* PABCO DECORATOR PRESERV WOOD CLEAR 1075

****

REGISTRANT *NAME AND ADDRESS*

* 009743 SKASOL INC
40 CLEVELAND ST
SAN FRANCISCO CA 94103

********** PRODUCT NAME **********

**00001* SKASOL BIOCIDE NO 2

**** PRODUCT SEARCH LISTING **** PAGE 92

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009754 GRO LYFE
DIV. OF SUNGRO CHEMICALS, INC.
810 E 18TH STREET
LOS ANGELES, CA 90021

********** PRODUCT NAME **********

**00004* WEED GO NON SELECT WEED AND GRASS KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 009803 ONEF II, INCORPORATED
875 INDUSTRIAL WAY
SPARKS, NEVADA 89431

********** PRODUCT NAME **********

**00005* MUNICHEM MUNI KILL

**00018* MUNICHEM POSITIVE WEED CONTROL LIQUID WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 009861 TECHNICAL SPECIALTIES CORPORATION
114 S COLUMBIA DR
DECATUR GA 30030

********** PRODUCT NAME **********

**00002* AQUA TECH TSC 902 ALGAECIDE BRIQUETTES

**** PRODUCT SEARCH LISTING **** PAGE 93

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009931 HANCO MANUFACTURING COMPANY
PO BOX 4476
MEMPHIS TN 38104

********** PRODUCT NAME **********

**00024* HANOBIGHT

**00025* 880 WEED SPRAY

**00030* FORESTAL WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 009957 STERLING CLARK LURTON CORP
184 COMMERCIAL ST
MALDEN MA 02148

********** PRODUCT NAME **********

**00001* STERLING PENTA DIP REDWOOD STAIN & PRESERVATIVE

**00003* STERLING PENTA DIP CLEAR NON STAINING WOOD PRESERVATICE

****

REGISTRANT *NAME AND ADDRESS*

* 009964 HANLINE BROS INC
1430 WARNER ST
BALTIMORE MD 21230

********** PRODUCT NAME **********

**00001* H&H PRODUCTS PENTALIFE WOOD PRESERVATIVE CLEAR

**** PRODUCT SEARCH LISTING **** PAGE 94

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010088 ATHEA LABORATORIES INC
4180 N FIRST ST
MILWAUKEE WI 53212

********** PRODUCT NAME **********

**00002* NON SELECTIVE LIQUID WEED KILLER

**00027* LWK 98 LIQUID WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 010119 JACKSON OIL PRODUCTS COMPANY
PO BOX 5686
JACKSON MS 39208

********** PRODUCT NAME **********

**00001* PENTA TREET READY TO USE WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011133 NATIONAL CHEMICAL
DIV OF SELIG CHEM IND 840 SELIG DR SW
ATLANTA GA 30336

********** PRODUCT NAME **********

**00002* ZEOLITE ALGAE BRIQUETTES FOR AIR CONDITIONING COOLING TOWERS

**00009* ZEOLITE NP 171 L ALGAECIDE

**** PRODUCT SEARCH LISTING **** PAGE 95

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010145 BLUMBERG COMPANY INC
PO BOX 566
PEABODY MA 01960

********** PRODUCT NAME **********

**00002* VITA SAN 75

****

REGISTRANT *NAME AND ADDRESS*

* 010148 NATIONWIDE CHEMICAL COMPANY INC
56 N 1ST STREET
BROOKLYN NY 11211

********** PRODUCT NAME **********

**00008* COMPLETE VEGETATION KILLER

**00009* SUPER VEGETATION KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 011155 GLOBE CHEMICALS INC
45 BURBANK DR
TOLEDO OH 43607

********** PRODUCT NAME **********

**00003* COMPLETE VEGETATION KILLER

**** PRODUCT SEARCH LISTING **** PAGE 96

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 01169 WALBICK COMPANY
9237 9TH AVE NW
SEATTLE WA 98107

********** PRODUCT NAME **********

**00001* KL 59 PENTA VAR SEALER

****

REGISTRANT *NAME AND ADDRESS*

* 01238 ZIMMITE CORPORATION
810 SHARON DRIVE
WESTLAKE OH 44145

********** PRODUCT NAME **********

**00001* CHEMTROL NO. 7 MICROORGANISM CONTROL CHEMICAL

****

REGISTRANT *NAME AND ADDRESS*

* 01292 VENUS LABORATORIES, INC
855 LIVELY BLVD
WOOD DALE, IL 60191

********** PRODUCT NAME **********

**00007* STA PUT 1200 WEED KILLER

*** PRODUCT SEARCH LISTING *** PAGE 97

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010383 WECO CHEMICAL COMPANY INC
BOX 123
GLEN ELLYN IL 60137

********** PRODUCT NAME **********

**00001* WECO WEED KILLER 104

****

REGISTRANT *NAME AND ADDRESS*

* 010413 SONFORD CHEMICAL COMPANY
8433 KATY FREEWAY
HOUSTON, TX 77024

********** PRODUCT NAME **********

**00001* SONFORD PENTA 5 READY TO USE SURE TREAT WOOD PROTECTOR

**00002* SONFORD PENTA 10:1 SURE TREAT WOOD PROTECTOR

**00003* SONFORD WOOD TREAT TECHNICAL PENTA CRYSTALS

**00004* PRIL TOX TECHNICAL PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010445 WATER MGMT DIV CALGON CORP
ATTN LEO HANEY CALGON CENTER BOX 1346
PITTSBURGH PA 15230

********** PRODUCT NAME **********

**00005* H 607 MICROBIOCIDE

**** PRODUCT SEARCH LISTING **** PAGE 98

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010485 UNITED CHEMICAL CORPORATION OF NEW MEXICO
PO BOX 1499
HOBBS NM 88240

********** PRODUCT NAME **********

**00017* ALPHA 542

**00018* ALPHA 541

**00019* ALPHA 540

****

REGISTRANT *NAME AND ADDRESS*

* 011599 RICE CHEMICAL COMPANY INC
2718 HAWKEYE DR
SIOUX CITY IA 51105

********** PRODUCT NAME **********

**00001* ALGAETRET=100

****

REGISTRANT *NAME AND ADDRESS*

* 011771 ADAMS ENGINEERING INC
P. O. BOX 3
STACY MN 55079

********** PRODUCT NAME **********

**00002* PATOX POLE TREATING BANDAGE I

**00003* PATOX POLE TREATING BANDAGE TYPE III

**** PRODUCT SEARCH LISTING **** PAGE 99

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010840 FARWELL OZMUN KIRK COMPANY
GILT EDGE PAINT MFG DIV 106 W FILMORE
ST PAUL MN 55107

********** PRODUCT NAME **********

**00001* GILT EDGE INTERIOR EXTERIOR WOOD SEALER

****

REGISTRANT *NAME AND ADDRESS*

* 010856 EVANS PRODUCTS CO
PAINT DIV
P.O. BOX 4098
ROANOKE VA 24015

********** PRODUCT NAME **********

**00001* GROSSMAN'S PENTALIFE WOOD PRESERVATIVE EXTERIOR CLEAR 47000

****

REGISTRANT *NAME AND ADDRESS*

* 011864 GAMIEN CHEMICAL COMPANY
4 MIDLAND AVE
E PATERSON NJ 07407

********** PRODUCT NAME **********

**00009* GAMACIDE 1831

**** PRODUCT SEARCH LISTING **** PAGE 100

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT	*NAME AND ADDRESS*
* 010867	WATER SERVICES INC P O BOX 10126 KNOXVILLE TN 37919

********** PRODUCT NAME **********

**00007* BAF KCN

**00008* BAF CIDE P

****

REGISTRANT	*NAME AND ADDRESS*
* 011880	MARCH CHEM COMPANY INC 1464 SIERRA VISTA DR BATON ROUGE LA 70815

********** PRODUCT NAME **********

**00001* MARCIDE 15

****

REGISTRANT	*NAME AND ADDRESS*
* 010932	AQUA SERV ENGINEERS INC 2431 E 56TH ST LOS ANGELES CA 90058

********** PRODUCT NAME **********

**00001* A 101 ALGAECIDE BRIQUETTES

**00003* A 103 LIQUID ALGAECIDE

**** PRODUCT SEARCH LISTING **** PAGE 101

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011291 PARKS CORPORATION
PO BOX 5
SOMERSET MA 02726

********** PRODUCT NAME **********

**00001* PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011482 BITTER LAKE CHEMICAL COMPANY
PO BOX 7528
SEATTLE WA 98133

********** PRODUCT NAME **********

**00001* VEGETOX BITTER LAKE ALL VEGETATION KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 011511 SHAICO CHEMICAL CORPORATION
2421 LEXINGTON AVENUE
TOLEDO OH 43606

********** PRODUCT NAME **********

**00001* SHALCO COMPLETE VEGETATION KILLER

**** PRODUCT SEARCH LISTING **** PAGE 102

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011528 ARMSTRONG CHEMICAL COMPANY
PO BOX 669
HUGO OK 74743

********** PRODUCT NAME **********

**00001* STERI KILL

****

REGISTRANT *NAME AND ADDRESS*

* 011547 SHARE CORPORATION
7821 N. FAULKNER RD P.O. BOX 2353
MILWAUKEE, WI. 53223

********** PRODUCT NAME **********

**00001* VEGETATION CONTROL WITH 2,4 D WITH DRIFT CONTROL ADDITIVES

**00002* VEGETATION CONTROL WITHOUT 2,4 D WITH DRIFT CONTROL ADDITIVES

**00015* WOOD PRESERVER

**00030* VEGETATION & SPOT WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 011576 IND WATER CHEMICAL DIV OF CONSOL FOODS
314 SPEARS AVE
CHATTANOOGA TN 37405

********** PRODUCT NAME **********

**00002* ALGACIDE L

**00003* ALGACIDE B

**** PRODUCT SEARCH LISTING **** PAGE 103

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011617 INDUSTRIAL WATER MGMT CORP
910 OXFORD CT
PALATINE IL 60067

********** PRODUCT NAME **********

**000004* A 1 ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 011655 SAMPSON PAINT MANUFACTURING COMPANY INC
BOX 4273
RICHMOND VA 23224

********** PRODUCT NAME **********

**000002* SAMPSON PENTA WOOD TREAT

****

REGISTRANT *NAME AND ADDRESS*

* 011668 T & R CHEMICALS, INC.
700 CEKUM ROAD
CLINT, TX 79836

********** PRODUCT NAME **********

**000002* T&R BRAND PENTA

**** PRODUCT SEARCH LISTING **** PAGE 104

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011682 SIMPLOT JR COMPANY MINERALS & CHEM DIV
BOX 810
MOUNTAIN HOME ID 83647

********** PRODUCT NAME **********

**00004* SIM CHEM PENTA 10.1 CONCENTRATE WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011690 AMERICAN SEAL MANUFACTURING COMPANY INC
669 RIVER ST
TROY NY 12180

********** PRODUCT NAME **********

**00002* PENTA PLUS CLEAR WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011694 DYMON, INC
3401 KANSAS AVENUE
KANSAS CITY, KS 66106

********** PRODUCT NAME **********

**00010* BKL SPRAY

**00018* BROMENOL 3 LIQUID WEED KILLER

**00043* BROMENOL 5

```
                    **** PRODUCT SEARCH LISTING ****                    PAGE     105

10/02/78        FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

*****************************************************************************

  *REGISTRANT*        *NAME AND ADDRESS*

  *  011712        KOR CHEM INC
                   1355 ELLISWORTH IND DRIVE N W
                   ATLANTA, GA 30318

    *************** PRODUCT NAME ***************

   **00007*  RUSTROL FORM 219B ALGAE BAGS

*****************************************************************************

  *REGISTRANT*        *NAME AND ADDRESS*

  *  011743        GUARDIAN IPCO INC
                   1315 2ND AVE NORTH
                   BIRMINGHAM AL 35201

    *************** PRODUCT NAME ***************

   **00001*  VULCAN #81 ALGAECIDE

   **00002*  VULCAN #85 ALGAECIDE

*****************************************************************************

  *REGISTRANT*        *NAME AND ADDRESS*

  *  012016        ANDERSON STOLZ CORP
                   1727 33 WALNUT ST
                   KANSAS CITY MO 64108

    *************** PRODUCT NAME ***************

   **00011*  SOL VET 302
```

**** PRODUCT SEARCH LISTING **** PAGE 106

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 013680 OZARK CHEMICAL COMPANY
PO BOX 1348
LITTLE ROCK AR 72203

********** PRODUCT NAME **********

**00002* PEAK #475 A NON SELECTIVE WEED AND BRUSH KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 013900 DELTA WATER LABORATORIES
4206 QUIRT AVENUE
LUBBOCK TX 79404

********** PRODUCT NAME **********

**00003* DELTA K 160

**00004* DELTA K 151

****

REGISTRANT *NAME AND ADDRESS*

* 014807 MARVELITE INC
3020 NIEMAN AVE
BALTIMORE MD 21230

********** PRODUCT NAME **********

**00001* SCOTLUX PENTALIFE WOOD PRESERVATIVE CLEAR

**** PRODUCT SEARCH LISTING **** PAGE 107

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 14886 PARKER ENGINEERED CHEMICALS INC
3613 CLEARVIEW PARKWAY NORTH PARK
ATLANTA GA 30340

********** PRODUCT NAME **********

**00004* P 22 ALGAE CONTROL TABLETS

****

REGISTRANT *NAME AND ADDRESS*

* 015146 CHEMWOOD CORPORATION
P.O. BOX 9158
MEMPHIS, TN 38109

********** PRODUCT NAME **********

**00001* CHEMTOX 10

**00002* CHEMTOX I

**00003* CHEMTOX S

**00005* POL GREASE

**00006* BUCHANAN SPECIAL BLEND

**00007* CHEMTOX PWR

**00008* CHEMTOX P WP CONCENTRATE

**00009* CHEMTOX P WR CONCENTRATE

**00010* CHEMTOX 7

**** PRODUCT SEARCH LISTING **** PAGE 108

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 015300 CHEMICAL TREATMENT COMPANY
500 LICKING HOLE RD HANOVER IND AIR PARK
ASHLAND VA 23005

********** PRODUCT NAME **********

**00004* CHEMICAL TREATMENT CL 208

**00005* CHEMICAL TREATMENT CL 205

****

REGISTRANT *NAME AND ADDRESS*

* 017664 BARCID DIVISION
N L INDUSTRIES INC 2404 S W FREEWAY
HOUSTON TX 77006

********** PRODUCT NAME **********

**00002* BAROCHEM B 460

****

REGISTRANT *NAME AND ADDRESS*

* 017866 INDUSTRIAL MAINTENANCE CORP.
3520 TRYCLAN DR
CHARLOTTE NC 28210

********** PRODUCT NAME **********

**00002* A 101 ALGAECIDE

**** PRODUCT SEARCH LISTING **** PAGE 109

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 018960 BASCO PRODUCTS COMPANY
P O BOX 142 HGHWY 49 SO
CLARKSDALE MS 38614

********** PRODUCT NAME **********

**00002* EVERGARD 500 PRIDE O MISS

**00003* BRUSH KILLER & GENERAL WEED KILLER CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 020375 NUTMEG CHEMICAL COMPANY
125 MARKET ST
NEW HAVEN CT 06513

********** PRODUCT NAME **********

**00011* NUTMEG NC 150

****

REGISTRANT *NAME AND ADDRESS*

* 023553 CATCMANCE LTD
94 BRIDGE RD E
WELWYN GARDEN CITY AL7 1JW UNITED KINGDOM

********** PRODUCT NAME **********

**00001* MYSTOX LEL 100%

**** PRODUCT SEARCH LISTING **** PAGE 110

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 025014 ENTERPRISE PAINT COMPANY
1191 SOUTH WHEELING ROAD
WHEELING, IL 60090

********** PRODUCT NAME **********

**00001* ENTERPRISE WOOD PRESERV WATER REPEL PENTA PROTECT WOOD 2 26

****

REGISTRANT *NAME AND ADDRESS*

* 026884 BONCO MANUFACTURING CORP
PO BOX 521
JEFFERSON GA 30549

********** PRODUCT NAME **********

**00003* BONCO ALGAECIDE BRIQUETTES

****

REGISTRANT *NAME AND ADDRESS*

* 030940 QUANTEX CORPORATION THE
BOX 9158 MALLORY STATION
MEMPHIS, TN 38109

********** PRODUCT NAME **********

**00001* QUANCIDE 3 FUNGIDE CONCENTRATE

**00002* QUANCIDE 2 FUNGICIDE CONCENTRATE

**** PRODUCT SEARCH LISTING **** PAGE 111

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 03`948 BIONOMICAL CHEM & SERVICE INC
1003 PINEVILLE RD
CHATTANOOGA TN 374^5

********** PRODUCT NAME **********

**00014* P 801 GENERAL WEED KILLER CONCENTRATE

**00017* PB 1^1 READY TO USE LIQUID WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 031969 BENFIELD INDUSTRIES, INC.
104 RICHLAND ST PO BOX 97
HAZELWOOD, NC 28738

********** PRODUCT NAME **********

**00002* SOUTHERN PINE WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* ^32235 NORCHEM COMPANY
PO BOX 2772
EUGENE OR 9740^2

********** PRODUCT NAME **********

**00001* END GROW VEGETATION CONTROL WITH DRIFT CONTROL ADDITIVE

**** PRODUCT SEARCH LISTING **** PAGE 112

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 032273 BEHR PROCESS CORPORATION
1603 W ALTON ST
SANTA ANA CA 92702

********** PRODUCT NAME **********

**00001* BEHR WOOD PRESERVATIVE NC 91

****

REGISTRANT *NAME AND ADDRESS*

* 032447 WOODHILL CHEMICAL SALES CORP
PO BOX 7183
CLEVELAND OH 45750

********** PRODUCT NAME **********

**00001* DURO WOODHILL WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 033283 BRANCHEMCO INC
8286 WESTERN WAY CIRCLE
JACKSONVILLE FL 32216

********** PRODUCT NAME **********

**00001* BRANCHEMCO 313

**** PRODUCT SEARCH LISTING **** PAGE 113

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 033355 SOUTHEASTERN LABORATORIES INC
117 WEST AVE
AYDEN, NC 28513

********** PRODUCT NAME **********

**00003* H 1 ALGAECIDE

****

REGISTRANT *NAME AND ADDRESS*

* 033404 STAT ENTERPRISES
1865 NEW HWY
FARMINGDALE, NY 11725

********** PRODUCT NAME **********

**00005* BULLS EYE LIQUID WEED KILLER WITH DRIFT CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 033560 PRO SERVE INC
P. O. BOX 161059 (400 EAST BROOKS RD)
MEMPHIS, TN 38116

********** PRODUCT NAME **********

**00001* STAA CLEAR WR

**00002* STAA CLEAN RTU FORMULA

**00003* STA CLEAR CONCENTRATE 1 10

**00004* STA CLEAR 10 S

**00005* STAA CLEAR WR 1 5

**** PRODUCT SEARCH LISTING **** PAGE 114

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 033576 CLIN WATER SERVICES
CLIN CORPORATION
120 LONG RIDGE RD
STAMFORD, CT 06904

********** PRODUCT NAME **********

**00001* S 531 B

**00009* S 533P

**00016* OLIN LIQUID ALGICIDE #C

**00017* OLIN ALGICIDE #3

**00021* MEMPO ALGICIDE #1

**00025* OLIN ALGICIDE 23

****

REGISTRANT *NAME AND ADDRESS*

* 034276 CENTURY INDUSTRIES CORP
RD #1
NEW WATERFORD, OH 44445

********** PRODUCT NAME **********

**00001* 5% PENTA WOOD PRESERVER

****

REGISTRANT *NAME AND ADDRESS*

* 034358 SIMONSEN CHEMICAL1COMPANY
P O DRAWER C
CABOOL, MO 65689

********** PRODUCT NAME **********

*** PRODUCT SEARCH LISTING *** PAGE 115

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

**CONTINUE REGISTRANT 034358

**00001* PENTA WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 034858 ALLADIN LABORATORIES INC
30 S OCEAN AVE BOX 569
FREEPORT, NY 11520

********** PRODUCT NAME **********

**00004* ALLADIN ALGICIDE PELLETS

**00006* ALLADIN ALGAE AND FUNGUS TREATMENT (FORMULA'G')

****

REGISTRANT *NAME AND ADDRESS*

* 036423 * **********

********** PRODUCT NAME **********

**00001* WOOD VAC

****

REGISTRANT *NAME AND ADDRESS*

* 036641 TRIANGLE PAINT CO., INC.
2222 THIRD STREET
BERKELEY, CA 94710

********** PRODUCT NAME **********

**00001* WOOD TREATMENT #174 CLEAR

**** PRODUCT SEARCH LISTING **** PAGE 116

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 037648 SOUTHERN IND. SOLES CORP.
WATER GUARD DIV
607 PARK AVE.
WILSON, N C 27893

********** PRODUCT NAME **********

**00004* BB 90

****

REGISTRANT *NAME AND ADDRESS*

* 039492 DEVOE & RAYNOLDS CO
DIV OF GROW CHEMICAL CORP
4000 DUPONT CIRCLE
LOUISVILLE, KY 40207

********** PRODUCT NAME **********

**00013* DEVOE ANTIFOULING MD 3260

**00021* DEVOE ANTIFOULING SHIP BOTTOM PAINT MD 3556

****

REGISTRANT *NAME AND ADDRESS*

* 039511 VERTAC, INC
STE. 3200 CLARK TOWER
5100 POPLAR AVE.
MEMPHIS, TN 38137

********** PRODUCT NAME **********

**00001* TECHNICAL PENTACHLOROPHENOL

*** PRODUCT SEARCH LISTING *** PAGE 117

10/02/78 FEDERALLY REGISTERED PRODUCTS CONTAINING PENTACHLOROPHENOL

REGISTRANT	*NAME AND ADDRESS*
* 039752	* *********

********** PRODUCT NAME **********

**00003* ELCO ALLCUT LIQUID WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 1

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 000909 COOKE LABORATORY PRODUCTS
4759 S DURFEE AVE
PICO RIVERA CA 90660

********** PRODUCT NAME **********

**04708* COOKE QUICK ACTION WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 001022 CHAPMAN CHEMICAL COMPANY
BOX 9158
MEMPHIS TN 38109

********** PRODUCT NAME **********

**05401* TIMPREG I

****

REGISTRANT *NAME AND ADDRESS*

* 002339 BAYER PRODUCTS LTD
KINGSWAY
LONDON, ENGLAND 00000

********** PRODUCT NAME **********

**09318* PENTA CURE

*** PRODUCT SEARCH LISTING *** PAGE 2

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 002439 APPERTON CHEMICAL COMPANY
PO BOX 2555 2903 STRICKLAND ST
JACKSONVILLE FL

********** PRODUCT NAME **********

**05661* PENTA CUBE

**09315* EX STAIN

****

REGISTRANT *NAME AND ADDRESS*

* 002553 BUHI CHEMICAL COMPANY
601 A BROOKHAVEN DR
ORLANDO, FL 32803

********** PRODUCT NAME **********

**04913* D & B PENTACHLOROPHENOL WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 003286 FERD STAFFEL CO.
P O BOX 238
SAN ANTONIO TX 78298

********** PRODUCT NAME **********

**08744* STAFFEL'S TERMI PHENE FOR GROUND AND WOOD TREATMENT

**08061* STAFFEL'S RIDALL BARE GROUND WEED AND GRASS KILLER

**** PRODUCT SEARCH LISTING **** PAGE 3

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 003579 BREWER CHEMICAL CORPORATION
P O BOX 48
HONOLULU, HI 96810

********** PRODUCT NAME **********

**03282* ULTRAMAR WR PENTA

**03284* NO 140 CONCENTRATE WOODTOX

****

REGISTRANT *NAME AND ADDRESS*

* 004652 CROWN CHEMICAL COMPANY
1416 N DIXIE HWY
HOLLYWOOD FL 33020

********** PRODUCT NAME **********

**09479* PENTA WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 004981 REDWOOD CHEMICAL INC
1215 JACKSON ST
HOUSTON, TX 77045

********** PRODUCT NAME **********

**04941* PENTA PLUS 40 WOOD PRESERVATIVE

*** PRODUCT SEARCH LISTING *** PAGE 4

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005302 LAWTON BROTHERS
2305 N ORANGE AVE
ORLANDO FL 32800

********** PRODUCT NAME **********

**05462* LAWOTN BROS 3 D WEED,VINE&BRUSH KILLER NON SELECTIVE WITH

****

REGISTRANT *NAME AND ADDRESS*

* 005660 FLORASTEEP CO
2810 OMEGA PLACE
ROSLYN PA

********** PRODUCT NAME **********

**09154* PENTA REGULAR

****

REGISTRANT *NAME AND ADDRESS*

* 005666 MAR VON CHEM COMPANY
CONGRESS PKWY
ATHENS TN 37303

********** PRODUCT NAME **********

**09157* PENTA WATER REPELLANT

**09158* MAR VCN CONCENTRATE PENTA 1 10

**** PRODUCT SEARCH LISTING **** PAGE 5

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 005967 MOYER CHEMICAL COMPANY
BOX 945
SAN JOSE CA 95108

********** PRODUCT NAME **********

**05146* PENTA 40

****

REGISTRANT *NAME AND ADDRESS*

* 006020 MOM CHEMICAL CO INC
2401 NORTH GREGG
MIAMI, FL 72701

********** PRODUCT NAME **********

**03375* INSTINCT TOTAL VEGETATION KILLER AND SOIL STERILANT

****

REGISTRANT *NAME AND ADDRESS*

* 006512 ADMIRALTY MFG COMPANY
PO BOX 631
SAN ANSELMO CA 94960

********** PRODUCT NAME **********

**10142* PENTAWOOD #77

**10143* PERMAWOOD #99

*** PRODUCT SEARCH LISTING **** PAGE 6

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 006720 SOUTHERN MILL CREEK PRODUCTS COMPANY INC
POX 1096
TAMPA FL 33601

********** PRODUCT NAME **********

**03358* SMCP "10 TO 1" PENTA

****

REGISTRANT *NAME AND ADDRESS*

* 006973 SOILSERV INC
PO BOX 1817
SALINAS CA 93901

********** PRODUCT NAME **********

**03573* SOILSERV PENTA 2

****

REGISTRANT *NAME AND ADDRESS*

* 007109 ST LOUIS PAINT MANUFACTURING COMPANY
2500 OHIO AVE
ST LOUIS MO 63104

********** PRODUCT NAME **********

**06806* MASTER SERVICE EXTERIOR WOOD STAIN WITH PENTZ

**06807* PANDA EXTERIOR WOOD STAIN WITH PENTZ

**** PRODUCT SEARCH LISTING **** PAGE 7

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 007358 DEL ROY PRODUCTS
823 LAGRANGE ST
TOLEDO OH 43609

********** PRODUCT NAME **********

**09841* RID MITE

****

REGISTRANT *NAME AND ADDRESS*

* 007478 CHEM PAK COMPANY
P.O.BOX 430757
SO MIAMI FLA 33143

********** PRODUCT NAME **********

**07981* TREET WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 007587 DRAKE CHEMICAL COMPANY
WEST RIVER ROAD
PERRYSBURG OH 43551

********** PRODUCT NAME **********

**09121* DRAKE PENTA WEED KILLER

*** PRODUCT SEARCH LISTING *** PAGE 8

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 008127 AGGIE CHEMICAL INDUSTRY
PO BOX 8335
SAN ANTONIO TX 78208

********** PRODUCT NAME **********

**05576* LAWN EDGER

****

REGISTRANT *NAME AND ADDRESS*

* 008278 METRO BIOLOGICAL LAB
8241 GAY ST
CYPRESS CA 90639

********** PRODUCT NAME **********

**10215* METRO(TESTED) SOILIZER NEW WEED KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 009319 CUSTOM CHEMICALS, INC
476 HESTER STREET
SAN LEANDRO, CA 94577

********** PRODUCT NAME **********

**04538* PENTASOL

**04539* PENTASOL 10 1

**04546* CUSTOM WEEDICIDE

**** PRODUCT SEARCH LISTING **** PAGE 9

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 009420 WATER CHEMISTS INC
1275 S BOYLE AVE
LOS ANGELES CA 90023

********** PRODUCT NAME **********

**07183* ALGICIDE 10 1L

****

REGISTRANT *NAME AND ADDRESS*

* 009782 WOODBURY CHEMICAL COMPANY
PO BOX 4319
PRINCETON FL 33030

********** PRODUCT NAME **********

**03648* PENTA 10 1WOOD PRESERVATIVE

**03650* PENTONE

****

REGISTRANT *NAME AND ADDRESS*

* 01395 BARCO CHEMICAL DIVISION INC
14800 N W 24TH COURT
OPA LOCHA, FL 33054

********** PRODUCT NAME **********

**04951* NON TOX NO 2

**** PRODUCT SEARCH LISTING **** PAGE 1

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 01569 J & P PETROLEUM PRODUCTS INC
PO BOX 4206 STA A
DALLAS TX 75208

********** PRODUCT NAME **********

**04404* TEX MASTIC PENTA WOOD PRESERVATIVE CODE NO. 510

****

REGISTRANT *NAME AND ADDRESS*

* 01727 MONSEY PRODUCTS COMPANY
COLDSTREAM RD
KIMBERTON PA 19442

********** PRODUCT NAME **********

**09018* MONSEY & GUARDSEAL PENTACHLOROPHENOL WOOD PRESERVATIVE

**09020* GUARDSEAL PENTA CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 010827 INDUSTRIAL SOLVENTS
PO BOX 312
SAN MARCOS TX 78666

********** PRODUCT NAME **********

**03429* PENTOCIL

**** PRODUCT SEARCH LISTING **** PAGE 11

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 010919 ACTEEN PEST CONTROL SERVICES
1011 HEINZ AVE
BERKELEY CA 94710

********** PRODUCT NAME **********

**05618* ACTEEN STOPS W D O

****

REGISTRANT *NAME AND ADDRESS*

* 011093 MASTER NURSERYMEN'S ASSN C/O LEO DUPUICH
3620 1/2 MT DIABLO BLVD
LAFAYETTE CA 94549

********** PRODUCT NAME **********

**07134* 49'ER GOLD STRIKE WEED KILLER 2,4 D PLUS DANVEL D

****

REGISTRANT *NAME AND ADDRESS*

* 011112 NOX WEED INC
6842 E LIVE OAK ST
BELL GARDENS CA 90202

********** PRODUCT NAME **********

**09639* NOXCIDE VEGETATION KILLER

*** PRODUCT SEARCH LISTING *** PAGE 12

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011134 PEST CONTROL CHEMICALS INC
5852 S WESTERN AVE
LOS ANGELES CA 90047

********** PRODUCT NAME **********

**06018* PESTCO PENTACHLOROPHENOL SOLUTION

****

REGISTRANT *NAME AND ADDRESS*

* 011157 ROSE EXT COMPANY
626 POTRERO AVE
SAN FRANCISCO CA 94110

********** PRODUCT NAME **********

**05601* ROSE'S WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011214 TARGET CHEMICAL COMPANY
17717 STUDEBAKER RD
CERRITOS CA 90701

********** PRODUCT NAME **********

**08132* PENTA 5 WOOD PRESERVATIVE

**** PRODUCT SEARCH LISTING **** PAGE 13

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011335 SCHULTE PAINT & LACQUER MANUFACTURING COMPANY
645 E HOLLY AVE
ST LOUIS MO 63147

********** PRODUCT NAME **********

**06170* SCHULTE WATER REPELLENT PENTA A NON STAININ WOOD PRESERVATEIVE

**09468* SCHULTE WATER REPELLANT PENTA-A NON STAINING WOOD PRESERVATIVE

****

REGISTRANT *NAME AND ADDRESS*

* 011464 GASPRO INC
PO BOX 2454
HONOLULU HI 96804

********** PRODUCT NAME **********

**07443* WEEDKILLER OSA #15 (OIL TYPE)

**07448* WEEDKILLER OSA #5 (OIL TYPE)

****

REGISTRANT *NAME AND ADDRESS*

* 011589 PAN AMERICAN CHEMICAL COMPANY
BOX 6186
MIAMI FL 33123

********** PRODUCT NAME **********

**07396* TERMITEICIDE

**** PRODUCT SEARCH LISTING **** PAGE 14

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 011656 WESTERN FARM SERVICE INC SHELL CHEM COMPANY
1025 CONNECTICUT AVE STE 200
WASH DC 20036

********** PRODUCT NAME **********

**05759* DORMANT 79 SPRAY

****

REGISTRANT *NAME AND ADDRESS*

* 011853 BISCAYNE CHEMICAL LABORATORIES
PO BOX 307, BISCAYNE ANNEX
MIAMI FL 33152

********** PRODUCT NAME **********

**10356* PENETRATING SOLUTION OF PENTACHLOROPHENOL

**10357* 40% SOLUTION OF PENTACHLOROPHENOL CONCENTRATE

****

REGISTRANT *NAME AND ADDRESS*

* 012661 GATOR CHEMICAL INC
1161 BASKIN ROAD
LARGO, FL 33540

********** PRODUCT NAME **********

**04518* GATOR PENTACHLOROPHENOL

**** PRODUCT SEARCH LISTING **** PAGE 1

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 013437 DU COR CHEMICAL CORP
P O BOX 13298
ORLANDO, FL 32809

********** PRODUCT NAME **********

**03533* DU COR WEED STOP

****

REGISTRANT *NAME AND ADDRESS*

* 014107 M CHEM INC
NEW ORLEANS LA 70123

********** PRODUCT NAME **********

**09351* M CHEM 1010 WEED CONTROL

****

REGISTRANT *NAME AND ADDRESS*

* 021275 SPAULDING INC
1921 5TH AVE S
ST PETERSBURG FL 33733

********** PRODUCT NAME **********

**05543* X CEL KWIK KIL

*** PRODUCT SEARCH LISTING *** PAGE 16

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 034164 AMERICAN REFINING & MFG. CO
P O BOX 332
MIAMI, FL 33147

********** PRODUCT NAME **********

**09258* PYNOL DISINFECTANT

****

REGISTRANT *NAME AND ADDRESS*

* 035031 CATC CHEMICAL COMPANY
1728 MARY STR
HONOLULU, HI 96819

********** PRODUCT NAME **********

**05397* KLENZ TERMITE KILLER

****

REGISTRANT *NAME AND ADDRESS*

* 035034 DIVERSEY HAWAII LTD
98-115 KANEHAMEHA HIGHWAY
AIEA, HI 96701

********** PRODUCT NAME **********

**06810* PENTA COAT WOOD PRESERVATIVE

**06812* TERMITOX TERMITE SOLUTION

**** PRODUCT SEARCH LISTING **** PAGE 17

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 035037 CHEMI=PURE PRODUCTS LTD
1210 ARTESIAN STREET
HONOLULU, HI 96814

********** PRODUCT NAME **********

**04485* X TERM O

****

REGISTRANT *NAME AND ADDRESS*

* 035052 GARDEN EXCHANGE LTD
63 KILAUEA AVENUE
HILO, HI 96720

********** PRODUCT NAME **********

**05464* GX WEEDKILLER

**05467* GX PENTA WB R T U COLORLESS

****

REGISTRANT *NAME AND ADDRESS*

* 035053 HONOLULU WOOD TREATING CO
2290 ALAHAE PLACE
HONOLULU, HI 96819

********** PRODUCT NAME **********

**05202* TERMACIDE

**05213* WOLMAN WOOD PRESERVATIVE SOLUTION

*** PRODUCT SEARCH LISTING *** PAGE 18

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 035069 HAWAII PLANING MILL
380 KANEELEHUA AVENUE
HILO, HI 96720

********** PRODUCT NAME **********

**03148* PENTA WR

****

REGISTRANT *NAME AND ADDRESS*

* 035217 HILO FARMERØS EXCHANGE, LTD.
318 KINOOLE STREET
HILO, HI 96720

********** PRODUCT NAME **********

**03716* F X WEEDKILLER

****

REGISTRANT *NAME AND ADDRESS*

* 035279 D P I QUALITY PAINTS, INC
532 SOUTH MISSOURI
CLEARWATER, FL 33515

********** PRODUCT NAME **********

**09276* NOX RCT WOOD PRESERVATIVE (PENTA) 106

**** PRODUCT SEARCH LISTING **** PAGE 19

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 035519 ECONOCHEMA CORPORATION
2719 S.E. EVERETT RD
CAMAS, WA 98607

********** PRODUCT NAME **********

**10587* PACIFIC WOOD PRESERVATIVE 10 TO 1 PENTA CONCENTRATE

**10588* ATCO 1410 PENTA TEN TO ONE CONCENTRATE

**10589* 1405 PENTA WOOD PRESERVATIVE

**10590* PENTA CONCENTRATE

**10591* ECONOCHEMA PENTA 5505 PENTA WOOD PRESERVATIVE

**10592* ECONO PENT A SEAL PENTA

**10593* 5510 ECONO CON PENTA TEN TO ONE CONCENTRATE

**10594* ATCO 1412 PENTA PRESERV A SEAL

****

REGISTRANT *NAME AND ADDRESS*

* 035880 KLINGERR PAINT COMPANY, INC
5555 HARNISHCHEEGER RD., S W
CEDAR RAPIDS, IA 52404

********** PRODUCT NAME **********

**06347* WOOD PRESERVATIVE CONTAINING PENTACHLOROPHENOL

**** PRODUCT SEARCH LISTING **** PAGE 2

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 036230 FOX HARDWARE
19 E CENTER STREET
MADISONVILLE, KY 42431

********** PRODUCT NAME **********

**06093* RED DEVIL READY TO USE BULLETT WOOD TREATMENT

****

REGISTRANT *NAME AND ADDRESS*

* 036392 CHARGAR CORPORATION
1011 DIXWELL AVENUE
HAMDEN, CT 06514

********** PRODUCT NAME **********

**10093* FLOROK WOOD PRESERVATIVE PENTA

****

REGISTRANT *NAME AND ADDRESS*

* 036688 BIG BEE CHEMICAL AND SUPPLY CO
ROUTE 4 OLD CONYERS ROAD
STOCKBRIDGE, GA 30281

********** PRODUCT NAME **********

**08199* WEED KILLER

**** PRODUCT SEARCH LISTING **** PAGE 21

10/12/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 036715 PENTA POST & TREATING CO.
TUTTLE ROUTE
BLISS, ID 83314

********** PRODUCT NAME **********

**03518* RAVENSCROFTS PENTA CONCENTRATE

**03519* RAVNSCROFTS PENTA READY TO USE

****

REGISTRANT *NAME AND ADDRESS*

* 037347 UNI CHEM CORP OF FLORIDA
BOX 6336 2801 N.W 55TH CT
FT. LAUDERDALE, FL 33310

********** PRODUCT NAME **********

**03125* UNI CHEM WEED KILLER AND SEMI-SOIL STERILANT

****

REGISTRANT *NAME AND ADDRESS*

* 037797 BROC DUGAN COMPANY
2145 SCHUETZ RD
ST. LOUIS, MO. 63141

********** PRODUCT NAME **********

**08382* PENTA WOOD PRESERVATIVE

**** PRODUCT SEARCH LISTING **** PAGE 22

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT *NAME AND ADDRESS*

* 037804 CHEM SERV, INC
2931 W FAIRMONT
PHOENIX, AZ 85017

********** PRODUCT NAME **********

**08403* CHEM SERV 440

****

REGISTRANT *NAME AND ADDRESS*

* 037919 ROBERTS JUDSON LUMBER COMPANY
301 EAST 4TH STREET
SALEM, MO 65560

********** PRODUCT NAME **********

**09424* BLOXROT C

****

REGISTRANT *NAME AND ADDRESS*

* 038099 SCHAEFER CHEMICAL INC
BOX 1008
TORRANCE, CA 90505

********** PRODUCT NAME **********

**09415* SCHAEFER CHEMICAL NO. 31 ALGAECIDE

**** PRODUCT SEARCH LISTING **** PAGE 23

10/02/78 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING PENTACHLOROPHENOL

****

REGISTRANT	*NAME AND ADDRESS*
* 038415	NOX CRETE CHEMICALS, INC. BOX 3764 OMAHA, NB 68103

********** PRODUCT NAME **********

**10071* PENTATOX WATER REPELLENT WOOD PRESERVATIVE

[FR Doc. 78-28936 Filed 10-17-78; 8:45 am]

Advance Orders are now Being Accepted for Delivery in About 6 Weeks

CODE OF FEDERAL REGULATIONS

(Revised as of April 1, 1978)

Quantity	Volume	Price	Amount
_____	Title 26—Internal Revenue Part 1 (§§ 1.301 to 1.400)	$4.00	$_____
_____	Title 26—Internal Revenue Part 1 (§§ 1.401 to 1.500)	4.75	_____
_____	Title 26—Internal Revenue Part 1 (§§ 1.501 to 1.640)	4.75	_____
_____	Title 26—Internal Revenue (Part 600 to End)	3.00	_____
		Total Order	$_____

[*A Cumulative checklist of CFR issuances for 1978 appears in the first issue of the Federal Register each month under Title 1. In addition, a checklist of current CFR volumes, comprising a complete CFR set, appears each month in the LSA (List of CFR Sections Affected)*]

PLEASE DO NOT DETACH

MAIL ORDER FORM To:

Superintendent of Documents, Government Printing Office, Washington, D.C. 20402

Enclosed find $..... (check or money order) or charge to my Deposit Account No. .....

Please send me ..... copies of:

PLEASE FILL IN MAILING LABEL BELOW

Name .....

Street address .....

City and State ..... ZIP Code .....

FOR USE OF SUPT. DOCS.

----Enclosed.....

To be mailed

----later.....

----Subscription.....

Refund.....

Postage.....

Foreign Handling.....

FOR PROMPT SHIPMENT, PLEASE **PRINT OR TYPE** ADDRESS ON LABEL BELOW, INCLUDING **YOUR ZIP CODE**

SUPERINTENDENT OF DOCUMENTS
U.S. GOVERNMENT PRINTING OFFICE
WASHINGTON, D.C. 20402

OFFICIAL BUSINESS

POSTAGE AND FEES PAID
U.S. GOVERNMENT PRINTING OFFICE
375
SPECIAL FOURTH-CLASS RATE
BOOK

Name .....

Street address .....

City and State ..... ZIP Code .....